VOL. XLIX. No. 4.

ARCHIVES OF OPHTHALMOLOGY.

SOME THEORETICAL AND PRACTICAL POINTS IN REFRACTION WORK.[1]

BY ALEXANDER DUANE, M.D., NEW YORK.

IN this paper I have endeavored to bring together in succinct form some facts in physiological optics that bear on the theory of refraction as well as some practical points in the handling of refraction cases. To forestall criticism, I should add that the former part of the subject matter contains nothing that cannot be gleaned from well-known works on physiological optics (Hess, Gullstrand, and others); and that the latter part simply reiterates views that I have strongly expressed before and which long experience has convinced me to be correct.

CARDINAL POINTS.

Any clear conception of physical and physiological optics must be based on the cardinal points of Gauss. We all know that the focus of a lens system is the point where parallel incident rays meet after refraction, and probably most have a conception of the anterior focus as the point in front of a lens where parallel rays will meet if their direction is reversed. We also know that the strength of a lens is inversely proportional to its focal distance. But from what point is this focal distance

[1] Read before the Section on Ophthalmology, New York Academy of Medicine, March 15, 1920.

to be measured? Gauss showed that, corresponding to the two foci, anterior and posterior (A and F), there are two principal points, anterior and posterior (E and I), and that the anterior focal distance is the distance between A and E and the posterior focal distance the distance between F and I. Further it was shown that no matter what the number of refracting surfaces or the character of the refracting media, the first focal distance, AE, is to the second focal distance, IF, as the index of refraction of the first medium is to the index of refraction of the last.

Later investigators added to these four cardinal points of Gauss the two nodal points (K, L), distant from the principal points by an amount equal to the difference between the anterior and posterior focal distances.

It is evident that when the first medium and the last medium have the same refractive density, as in the case of a lens in air or of the crystalline lens in the eye, the anterior and posterior focal distances are equal and that consequently the nodal points coincide with the principal points.

The case is different with the eye as a whole. Here the first medium, air, has a refractive index of 1, the last medium, vitreous, has a refractive index of a little over $1\frac{1}{3}$. Hence the anterior focal distance being about 15.8*mm*, the posterior must be $15.8 \times 1\frac{1}{3}$ or 21.1*mm* and the nodal points must be $21.1 - 15.8 = 5.3$*mm* behind the principal points. The principal points, in fact, are just behind the cornea, the nodal points at the posterior pole of the lens.

The principal significance of the cardinal points is shown in the following statements:

1. The position of the posterior focus with relation to the retina determines whether the eye is emmetropic, hypermetropic, or myopic.

2. The position of the posterior nodal point with relation to the retina determines the size of the retinal image and has, therefore, a bearing on the apparent size of objects and on visual acuity.

3. The main effect of glasses placed in front of the eye is not to change the refractive power of the latter but to shift the position of the cardinal points—putting the posterior focus of the eye (F) on or off the retina, and changing the position of the posterior nodal point (K) with relation to the latter.

4. If a glass is placed at the anterior focus of the eye—and this is the regular place for correcting glasses—it makes absolutely no change in the refractive power of the eye. That is, a + 10 D. lens placed 14*mm* in front of the cornea has no effect whatever on the posterior focal distance of the eye which remains 21.1*mm*. What it does do is to shift the posterior focus of the eye and also the posterior nodal point of the eye some 3*mm* forward and thus exert a marked and determinate effect on the clearness of retinal images.

5. On account of this and other properties, the anterior focus of the eye is the point where we should place glasses to be worn, where we should place the ophthalmoscope in applying the direct method, and from which we should make our measurements in determining the far point and near point of accommodation.

EFFECT OF POSTERIOR SURFACE OF CORNEA ON THE REFRACTIVE POWER OF THE EYE.

Usually in calculating the constants of the eye, the effect of the posterior surface of the cornea is neglected. It is not, however, a negligible quantity. Thus if we use the latest measurements of Tscherning in calculating the constants of the eye, we find a posterior focal distance of 20.79 if we disregard the posterior corneal surface. Using the same constants and taking the effect of the posterior corneal surface into consideration I find the posterior focal distance to be 21.14. The difference corresponds to a difference of a whole dioptry in refraction.

STANDARD EYE NOT HYPEROPIC.

I should object to the idea that the eye which has standard measurements is hyperopic instead of emmetropic. Measurements vary a good deal but those of the best observers from Helmholtz down give for the position of the posterior focus of the eye a point 22.8 to 23.9*mm* behind the cornea. The variations probably range within even wider limits than these and, so too, does the axial length of the emmetropic eye, which may be anywhere from 22 to 25*mm*. In any event, the figures do not show that regularly or often the posterior focus of the unaccommodated normal eye falls behind the retina.

RÔLE OF THE PUPIL.

The pupil as we see it through the magnifying lens formed by the cornea is about one ninth larger than the actual pupil. But this apparent pupil ("Einstrittspupille" or "inlet" pupil as Abbe calls it) measures accurately the width of the beam of rays that can find its way through the actual pupil. That is, a pupil really 4.5*mm* in diameter, and which appears to us to be 5*mm* in diameter will admit a beam of rays that occupies a circle of the cornea that is 5*mm* in diameter.

Opposed to this "inlet" pupil is the "outlet" pupil ("Austrittspupille") or image of the pupil formed by the crystalline lens. It is this figure which really forms the base of the cone of rays directed toward the retina, and determines therefore the size of diffusion images. It is, however, very little larger than the pupil itself.

CHANGE OF REFRACTION IN DEVELOPMENT OF EYE.

I think we often get a wrong conception of the change that takes place in the refractive state of the eye between infancy and adult life. We think of it, that is, as a change caused by simple elongation of the eyeball converting a primitive axial hyperopia into emmetropia and in many cases into myopia. But a moment's consideration shows that this is a very partial representation of what takes place. The infant eye is so short that, built on anything like the refractive scheme of the adult, it would have an axial hyperopia of 20 D. As in reality it is usually hyperopic only 2 or 3 D. at most—and sometimes not hyperopic at all—it is evident that in the early years of life we have a combination of two factors—an axial shortening sufficient to cause a hyperopia of 20 D. and an excess of curvature sufficient to cause a myopia of some 18 D. This excess of curvature must be located in the lens, since the cornea is not very much more curved in the infant than in the adult. As the child grows, two opposing processes occur simultaneously. The excessive curvature of the lens diminishes, causing a reduction of the curvature myopia; and the eyeball elongates, causing a reduction of the axial hyperopia. Usually, but not invariably, the latter process predominates so that the eye as a whole

tends to grow less hyperopic—strives to become emmetropic or sensibly so. Under so-called civilized conditions this natural process is largely thwarted by circumstances of environment—excessive eye work and other disturbing factors.

This process of "emmetropization" has been especially emphasized by the Dutch observer Straub, who also called particular attention to its duplex character. Straub, however, as is the custom with some in Europe, grouped under emmetropia cases which here we should regard as latent hyperopia—*i.e.*, cases in which a hyperopia of a dioptry or more is habitually overcome by accommodation. Hence he was led to regard many as cases of complete emmetropization which we should not regard as such. Even so, it is rather remarkable that a process composed of two opposing and simultaneously diminishing factors should not present more variations than it does, and that there should be on the whole a steady progress toward emmetropia—even if this does often fall short of the mark or overshoot it.

ASTIGMATISM.

The researches of Gullstrand have shown that our conceptions of astigmatism must be very greatly modified. The familiar figures used in the text-books, showing Sturm's conoid and Sturm's interval and the diffusion images formed by a sphero-cylindrical surface give us only a rough idea of astigmatic refraction. This is true even of ordinary lenses and still more true of the astigmatic refraction of the eye which is a very complicated affair, indeed. Gullstrand shows that the diffusion images formed by a sphero-cylinder are not the regular ellipses and lines usually pictured, but are stellate figures. But he also points out that the shape and size of the diffusion image are not of so much consequence in determining the clearness of vision, as the way in which the light is concentrated in the diffusion image. If the main part of the light is massed in the center of the latter, the hazy area about this will be little in evidence, and we shall get a fairly distinct image. This, I may say, is evident even when we trace out the formation of images through a simple cylindrical lens by a simple mathematical analysis.

There are two points in connection with astigmatism upon which there has been some confusion.

The first concerns the astigmatism of the cornea as shown by the ophthalmometer in the central and in the peripheral areas. The use of the ophthalmometer is open to two limitations. The instrument really determines the radius of corneal curvature at the point of examination. It determines the astigmatism only by inference. That inference is based on two suppositions, first, that the refractive state of the various media behind the cornea is of a certain character; second, that there shall be no cause for the corneal astigmatism other than the unequal corneal curvature. The first supposition is violated when, without suitable correction, we apply the measurements of the ophthalmometer to the determination of astigmatism in the aphakic eye. The second is violated when in the ordinary eye we measure with the ophthalmometer corneal astigmatism at some distance from the optic axis. For in this outlying district of the cornea there is superadded to the astigmatism due to unequal corneal curvature, which the ophthalmometer shows, the astigmatism due to aberration, which the ophthalmometer does not show. I may illustrate this point as follows: If the cornea were perfectly spherical in curvature at its apex and throughout, the ophthalmometer, of course, would show no astigmatism no matter where applied. Yet, as calculations show, at 1.25*mm* from its center such a cornea would have 0.25 D. astigmatism due to aberration, and this astigmatism would increase *pari passu* as we traveled outward. This astigmatism the ophthalmometer would not reveal. On the other hand, if the cornea outside of the central area were so flattened as to do away with the astigmatism due to aberration, it would, though non-astigmatic, show an increasing corneal astigmatism by the ophthalmometer. These facts should lead us to modify any conclusions drawn from direct measurement of corneal astigmatism outside of the visual area, made with the ophthalmometer. Astigmatism, as thus measured, is sometimes greater, sometimes less than the true corneal astigmatism; and a certain amount as thus measured may really represent a compensation for astigmatism actually present.

The second point that I should like to make is this. It has

been stated that astigmatism cannot be adequately corrected by a cylinder placed at the anterior focus of the eye; the reason alleged for this statement being that any glass so placed does not alter the size of the retinal image, and therefore cannot relieve the distortion caused by the astigmatism. This view I hold is incorrect. It is true that, as already stated, such a glass does not alter the refractive power of the eye. But it does shift the focus of the eye in the astigmatic meridian. Now the essence of astigmatism is that the rays in one meridian (A) are focused in a point farther back than those in meridian (B) at right angles. If by a glass we bring the focus of the rays passing through A up to the focus of those passing through B we have overcome the astigmatism and produced a clear image. Moreover, we have reduced the size of the diffusion image formed by the rays passing through A to practically normal dimensions. If the statement that I am controverting is correct, it would mean that we could not change the quasi-linear astigmatic image formed by a sphero-cylinder to a punctate image by placing a correcting cylinder at some distance in front of the glass. But this we can do. Take a + 20 D. spherical lens combined with a + 4 D. cylinder axis 90°, and form with this the image of a moderately distant point of light. The image will be more or less linear. Place at the anterior focus of this artificial astigmatic eye, namely 5*cm* in front of it, a − 5 D. cylinder, axis vertical. The linear hazy image will be reduced to one that is sharp and punctate as if formed by a spherical lens.

LENS IN ACCOMMODATION.

Gullstrand by his exhaustive analysis has shown the character of refraction through the lens when at rest and in accommodation. I will simply say here that his researches furnish further proof of the essential validity of Helmholtz's theory of accommodation, and further demonstrate that, owing to its special structure, the lens in accommodation not only changes its shape but also becomes absolutely more refractive—that is, its total index of refraction is greater in accommodation than when at rest.

He also showed that the lens consisted of a series of isoindi-

cial refractive surfaces, *i.e.*, surfaces at all points of which the lens substance had equal indices of refraction. At the most exterior of these surfaces coincident at the poles of the lens with the exterior surface of the latter the index of refraction is 1.386. The isoindicial surface close to the center of the lens has an index of refraction of 1.404, while the very center has an index of refraction of 1.406. The isoindicial surfaces are ellipsoidal in form, the long axis of the ellipsoid being vertical. In accommodation these ellipsoids shorten vertically and spread out in a fore-and-aft direction so as to become more nearly spherical. This tendency is decidedly more marked in the central than in the peripheral isoindicial surface. In accommodation the lens increases 11 per cent. in thickness; the curvature of the anterior surface increases 87 per cent. and that of the posterior surface 12.5 per cent.

Hess and others have taught that the ciliary muscle has practically the same contractile power in childhood, youth, and middle life, so that it is able to put forth the same accommodative effort at one age as another. In this view the child of ten and the man of forty-five has each an accommodative power of, say, 15 D., the only difference being that in the child practically all this power can be exerted in expanding the lens, while in the man only a small portion of it can be so exerted, the rest being unused and latent. It would follow from this that if some agent was employed that would reduce the accommodation to 10 D., it would show its full effect in the child, whose near point would recede from 6.6 to 10*cm*, while in the man it would not be in evidence at all since all the accommodation that he needs at his eye is 4 D., and he still has 10 D. left. Even in a person of twenty, whose near point is at 10*cm*, the effect would be barely noticeable. This, in fact, is the inference that these authors make—their belief being that paralysis of accommodation, unless extreme, cannot be made out in middle life or beyond. But this is untrue. In homatropine we have an agent causing a gradually increasing, ultimately quite complete paralysis of the ciliary muscle. On Hess's theory homatropine ought to produce a noticeable effect very quickly in children, much later at the age of twenty or twenty-five, and very late, if at all, in persons over forty. But careful tests under conditions which exclude the effect caused by com-

plicating factors (*e.g.*, the dilatation of the pupil due to the drug) show that paralysis from homatropine becomes evident almost as quickly in the older patient as in the child. I have made an extensive series of observations on this subject which seem to show this conclusively.

SOME PRACTICAL POINTS IN THE CONDUCT OF REFRACTION WORK.

On most points connected with the practice of refraction we are all agreed. On some, notably on the question of the use or non-use of cycloplegics, there is still much difference of opinion.

As a contribution to the question I should like to offer my testimony based on my life's experience in refraction work.

1. **Cycloplegia Necessary in the Determination of Refractive Errors.**—In the great majority of cases the refraction of the eye cannot be determined with certainty without the use of cycloplegics. This statement applies to astigmatism as well as to hyperopia and to both more than it does to myopia.

In making this statement I would not for a moment be understood as underestimating the necessity of making a careful test of the eye in its natural condition. This I myself invariably do and in a very large number of cases by so doing arrive at the same result as under a cycloplegic. But as I can never be sure in any given instance that this will be the case and as in some cases in which I had reason to think that the results would be the same they have turned out to be different. I feel that I must give myself and the patient the added certainty that the use of the cycloplegic affords. At the same time I feel it necessary to make the most careful precycloplegic test in order to see what the eye can do and what it will accept under natural conditions. By comparing the result with that obtained under the cycloplegic I feel that I have full data on which to base a proper judgment as to the prescription to be given.

What, in fact, are the objections urged to the use of a cycloplegic?

They are these:

(*a*) A cycloplegic is useless, since the refraction can be de-

termined sufficiently well without it. But this is contrary to my experience. In many and many a case not only would I have failed to determine the full correction, but, what is more to the purpose, would have failed to determine the glass that gave the patient comfort, unless I had used the cycloplegic.

(*b*) The cycloplegic often fails to disclose the true refraction. But this, again, I cannot corroborate from my experience if cycloplegics are used in the proper strength and if their action is checked by the accommodation tests in the manner given below. Once in a while the tests are contradictory or unsatisfactory under homatropine. This may be because the range of accommodation remains too high. We discover this fact from our accommodation tests. In that case I use atropine. But this occurs quite exceptionally. In the great majority of cases homatropine has proved itself in my experience reliable, giving complete and satisfactory cycloplegia, although it may perhaps at times fail to disclose 0.25 D. of the hyperopia, which may be found on examination made two or three years later. But even here there is a question whether the refraction has not actually changed.

(*c*) A cycloplegic is dangerous because it may produce an acute glaucoma. This has happened to me once in my entire experience. The danger, in fact, is about as great as that of being injured in a railroad accident. We do not give up riding on a railroad because of the fear of being killed some day, if we do. Nor do we give up the use of a cycloplegic because once in ten or twenty thousand times the latter produces glaucoma.

Furthermore, an eye which develops glaucoma under a cycloplegic is an eye which is going to develop glaucoma anyhow. It is not an unmixed evil then if it develops it right before our eyes. We can usually control the increase of tension with eserine or, failing that, with a paracentesis, and then do the iridectomy that such a case should in any event have.

(*d*) We do not prescribe the glass we find under a cycloplegic, so why use this latter? By using a cycloplegic we have found the absolute static refraction of the eye. Whether we shall prescribe the glass fully correcting this or not is a question to be determined by various considerations, but at all events we have found the basic facts in the case on which to build our result by such addition or subtraction as seems proper. We

might as well say, "We are not going to give a presbyopic patient his distance glass to read with; why then find his distance glass at all?" But we know that, even if we are not going to give a patient any distance glass, it is necessary to determine with accuracy and thoroughness the exact amount of error in each eye for distance, and then using this as a foundation, determine the added correction that the patient needs for near.

In determining from the cycloplegic test the glass that a patient should wear, it is important to remember that the glass which gives the best vision for fifteen or twenty feet may have to be reduced by 0.25 D. in order to give really clear vision for distance. I often, in order to determine this point, ask the patient when under a cycloplegic to look out of the window at the details of a distant object and say whether these are accentuated by the addition of −0.25 D. If so, I consider that his static refraction is measured by the glass with this addition.

2. **Use of Cycloplegics after Forty.**—Contrary to what is often stated, I have found that it is even more necessary to use a cycloplegic in middle life than in youth. Those of middle life often relax the accommodation imperfectly or with difficulty, and are often rather resistant to cycloplegics. The more flexible ciliary muscle of youth usually relaxes more readily. Personally I use cycloplegic in practically all cases up to forty-eight, and sometimes after that age.

In a paper read before the New York Academy of Medicine in 1908, I cited nineteen cases of patients between forty and forty-nine in whom homatropine had disclosed latent hyperopia of anywhere from 0.62 to 3.00 D. and latent astigmatism of from 0.25 to 1.00 D. What was particularly significant was that sometimes considerably more error was latent in one eye than in the other, so that no conceivable equal addition to the manifest correction would have given a glass satisfactory for both eyes. This point is clearly brought out in the paper cited as well as the fact that in other respects, too, a satisfactory correction would have been quite unattainable in these elderly patients without the use of a cycloplegic.

3. **Determination of Effect of Cycloplegic.**—To determine whether or not we get complete cycloplegia we should systematically employ the accommodation tests, taking the ac-

commodation before and during the instillations until the range has been reduced to below a dioptry. This is done as follows:

Suppose we have a patient with a manifest error of + 1.75 + 1.25 cyl. 90°. Putting this correction on we measure his accommodation with the Prince's rule and the accommodation line. Suppose the accommodation to be 4.5 D. We instill the cycloplegic and measure repeatedly thereafter. The accommodation sinks first to 3.5 and 3 D., then below. At this point we add a convex sphere of 1 or 2 D. to the above combination and again test the accommodation. When finally with + 3.00 added the far point is found to be at 3 and the near point at 3.75, we know that the range of accommodation is reduced to 0.75 D. and that the patient is ready for examination. This period of complete cycloplegia occurs at a time varying anywhere from forty minutes to two hours or more. Tests taken when the accommodation is not thus completely relaxed are apt to give wrong results.

We further use the accommodation test as a check to the result. If our final finding is, say, + 2.25 + 1.25 cyl. 90° then with + 5.25 + 1.25 cyl. 90°, the patient's far point should be just 3 D. If it is we feel that our test is corroborated.

4. Precycloplegic and Postcycloplegic Tests.—As stated, I try to make as careful as possible a precycloplegic test. In so doing I often get help from an expedient which I first learned from Dr. C. H. May. When the manifest correction in each eye has been obtained and apparently no further relaxation of the accommodation can be secured, I put on the glasses found, direct the patient to look with both eyes and then add to this binocular correction convex glasses by successive increases of + 0.25 D. Patients who have refused to take any further additions in monocular vision, may, when looking with both eyes, be led to accept as much as a dioptry or more of convex correction. The addition is made to both eyes simultaneously until blurring begins; then the effect is tried of adding to or subtracting from one glass (the patient still looking with both eyes).

When there is a large disagreement between the precycloplegic and the cycloplegic findings, I insist on a postcycloplegic test. This should be made not less than four days after the cycloplegic, since some trace of the latter often persists for this

length of time, as shown by the accommodation tests, and if we make the test too soon we are likely to prescribe a glass that will be uncomfortably strong or one that will blur the distant vision. I prefer to make my postcycloplegic tests not less than a week after the cycloplegic, particularly when it is a question of giving a presbyopic glass based on the cycloplegic finding. Here we wish to be particularly careful that the full power of accommodation has returned.

5. **Necessity of Routine Accommodation Tests.**—Together with careful refractive measurement, precycloplegic, cycloplegic, and if necessary postcycloplegic, careful repeated tests should be made of the accommodation and muscles. Insufficiencies of accommodation are of great importance at all ages, though particularly in the young, and if they are not recognized and treated, the results of correction of the refraction may be disappointing. Contrary to the view expressed by Hess and others, insufficiency of accommodation is of significance and can be made out in persons of the presbyopic age.

Testing of the accommodation is of particular importance in ensuring proper presbyopic correction. Few patients of forty-eight or over will tolerate a reading glass that makes their near point closer than 3.5 D. Often, especially if they have a convergence insufficiency, a near point of 3.25 D. is the most they will bear. Others again show an unequal maximum of accommodation in the two eyes, *e.g.*, with a 2.50 addition show a near point of 3.50 D. in one and only 2.75 D. in the other. In this case often, but not invariably, we shall have to make allowance for the inequality, and give an addition of + 2.00 in one eye and + 2.25 or 2.50 in the other.

6. **Necessity of Careful Muscle Testing.**—So too, the determination of the muscle balance for far and especially for near points is of great significance in our refractive work. In the presence of a convergence excess we crowd our hyperopic correction. In the presence of a convergence insufficiency, evidenced by a marked exophoria for near and a remote convergence near point, we are careful not to give a strong presbyopic correction, which in just these cases is badly tolerated. In cases of hyperphoria we may have to add a vertical prism or in certain cases a prism paster to ensure comfortable vision. And sometimes the proper glasses can be made tolerable only after a

proper course of muscular training. Some cases can be made comfortable only by operation.

7. Factors governing the Treatment of a Refractive Error.—Treatment of our refraction cases should be based on the following considerations.

(*a*) The *age* of the patient. Obviously the younger the patient the more we can leave for his accommodation to correct. Nor must we forget that in children symptoms have not the same significance as in the adult. Oftentimes headache, asthenopia, etc., are transitory and depend on the effect on the sharply reacting nervous system of the child of various influences of which the refractive error is only one. Hence, unless there is some strong reason for giving glasses at once, I am inclined to refrain from prescribing them in the case of young children. Hyperopia of a dioptry or more and astigmatism of 0.50 D. at least may, I believe, be safely left uncorrected, the patient, however, being watched to see whether the symptoms do or do not persist under this temporizing policy. I have never had occasion to regret adopting this waiting plan, and by it have been able to save several children from the inconvenience of using glasses.

This remark, however, applies only to hyperopia. Here a temporizing plan is all the more justifiable since the hyperopia naturally tends to diminish as the child grows. But in myopia in a child I insist on the full correction at once, believing that this is the best means for preventing the increase of the nearsightedness.

(*b*) The *vision*. Patients who have sharp vision for distance and who make sharp distinctions—who can readily recognize distinctness in the strength and axis of added glasses—naturally require a particularly accurate correction. With such patients we shall be especially concerned to get the glass right to a 0.12 D., and in these a weak glass often affords a relief that we should not expect and that in other persons, less discriminating, we fail to obtain. On the other hand it is undeniable that others—and particularly those with reduced vision—often get on satisfactorily with an approximate correction. This is fortunate since in just these cases an approximate result is often all that we can get.

Certain people demand sharp vision and suffer real annoy-

ance unless they get it. In these cases we must be particularly careful not to give an over-correction of hyperopia or an under-correction of myopia for distance. Others, particularly myopes of medium degree (4 to 8 D.), are not only satisfied with a somewhat blurred vision for distance but actually prefer it saying that the sharp definition is actually disagreeable. Again, many in office work are willing to forego sharp distant vision if they can see distinctly at a yard or two. In such it is often best to give a bifocal, in which the upper part represents the distance glass with a + 1.00 D. added and the lower the regular reading correction.

Lastly, patients with reduced vision for near will usually require a somewhat stronger reading glass than those with sharp sight.

(*c*) *Amount and Kind of Use to which Eye is Put.*—Obviously a person who is engaged in hard continuous eye work under trying conditions needs correction more than one who is not. In this regard, we must remember, first, that some forms of distant vision, *e.g.*, looking at objects in a theater, museum, or stadium, using the eyes on a train or in a motor car, etc., are more trying even than near work; second, that some forms of near work are more trying than others. Many find card-playing more taxing to the eye than either reading or writing, and, in certain forms of close work (foreign language study, proof-reading, reference work of all kinds, fine embroidery, fine draughting, etc.) the demands made on the eye are such that the most particular correction of even slight degrees of error, especially astigmatism, is required. Similarly a patient who uses the eyes much by artificial light will require a correction where he might not otherwise do so; or, if a presbyope, will require a stronger addition than he would by daylight.

In considering what is and what is not trying eye work we must remember that to children of six or eight, reading of English means what reading of a foreign language does to an adult and they may require help from glasses for what would seem to us an easy task.

(*d*) *The Symptoms and their Obvious Relation to the Use of the Eyes.*—We give glasses not because a patient has this or that refractive error, but because he has this or that symptom or

because we feel assured that without glasses he is reasonably certain to develop trouble.

Again, no matter what the patient's symptoms are, we must, in prescribing glasses for their relief, be first fairly confident that the symptoms are, in part at least, either caused or aggravated by the eye condition.

In this regard we are likely to make two mistakes. First, we may consider as of ocular origin certain symptoms, such as eyeache and asthenopia, which though ocular in seat are really dependent on other causes, especially nasal obstruction or dental infection. I have repeatedly seen instances where correction of these extraocular conditions relieved symptoms which were apparently due to eye trouble and which were certainly induced by the use of the eyes. But while we should not fall into the mistake of regarding all eye symptoms as necessarily due to the eyes, we must equally avoid the opposite mistake of failing to recognize the influence of refractive, accommodative, and muscular errors of the eyes in causing all sorts of symptoms, some of them not apparently ocular. We must remember that an asthenopia of nasal origin will regularly be aggravated by the use of the eyes and will require help from glasses as well as nasal treatment. It is the mark of the skilled ophthalmologist as distinguished from the mere refractionist that he is able to discern the relative importance of all the factors, extraocular as well as ocular, and in some measure assign to each its probable effect.

We must also bear in mind the considerable difference between individuals with regard to their reaction to strain of any kind. Some seem to be able to stand all kind of stresses; others suffer intensely from slight abnormalities and their consequences. The latter sort of patients we must correct with extreme care and patience. Nor should we too lightly dismiss complaints that seem to us querulous and ill-founded. Slight blurring of vision, almost indefinable symptoms of discomfort, annoyances arising from maladjustment—difficulties which seem to us very trivial—may in sensitive individuals produce what is to them intolerable annoyance. These troubles are more real than we are apt to think. Patient attention to them and their correction and extreme care in the selection, adjustment, and verification of glasses are required and should be given.

(*e*) *The Accommodation* and (*f*) *Associated Muscular Conditions of the Eyes.*—As already stated careful attention should be paid to these points in every routine examination of the refraction, and proper steps should be taken to correct any outstanding defects which are giving symptoms. With regard to this point it may be said that it is often difficult in the presence of a muscular anomaly to tell whether this or the refractive error is the cause of the symptoms. In this case the refractive error should be corrected first, and after a reasonable interval given to ascertain what this alone will accomplish, measures should be taken to relieve if necessary the effects of the motor anomaly.

(*g*) *The Associated General Symptoms and General Condition of the Patient.*—The influence of the patient's general condition on his ability to do eye work is obvious. A patient depressed by overwork or in other ways may develop eye symptoms, the relief of which may require both treatment addressed directly to the eyes and treatment designed to remove the cause of the systemic trouble. Patients who are mentally depressed or mentally backward and patients who are nervous or supersensitive require special care in examination and special attention in treatment. The relief of their eye condition may be an essential element in their improvement and we must give our best efforts to secure good results in this regard. At the same time in our zeal to correct the eyes we must not forget the individual and his idiosyncrasies nor the fact that the correction of his other physical and his nervous ailments claim equal attention with the ocular treatment.

To give the patient the best service we may have to forego for a time the treatment of the eyes and call in the aid of other specialists or of a general practitioner.

I think the whole matter of handling refraction cases may be summed up in these two maxims:

First, let us find out all we can about the eyes we are treating and the symptoms of which they are a part; using to this end every means that experience has proved to be helpful, never doing the work in a hurry, and remembering always that this work of all others requires patience, thoroughness, and accuracy.

Second, let us constantly bear in mind the fact that we are

treating patients, not eyes; we are handling human beings, not machines; and that we cannot do our best work unless in each case we put ourselves in touch with the individual man before us, showing sympathy for his troubles, consideration for his infirmities, and an understanding mind to take in all the physical and mental factors which may affect his outlook on life and determine his need for refractive or other correction.

AFFECTIONS OF THE EYE FROM DISEASED TEETH IN THEIR RELATION TO THE LYMPHATIC SYSTEM.

By KAETHE W. DEWEY, M.D.

From the Research Laboratory of the College of Dentistry, University of Illinois.

RELATIONS between certain affections of the eye and diseased teeth have been recognized since the remotest ages, but only in relatively recent years have such views received the necessary scientific foundation through clinical and anatomical investigations. Although the number of reliable reports of actually observed transmissions of pathological processes from the teeth to the eye is not inconsiderable, little is yet known concerning the routes of transmission that could in any way be considered as positive and final. Probably all these transmitted processes are of a bacterial and bacteriotoxic nature. The dental origin of cases of the so-called dental eye fistula, orbital phlegmon, and abscesses is revealed chiefly through the fact that the eye conditions either promptly disappeared with treatment of the involved teeth, or that they developed upon the extraction of a tooth, with or without involvement of the maxillary sinus. The traveling of the pus from diseased teeth to the orbit, as for example in phlegmon, has often been represented as being *per continuitatem* along the outer surface of the maxilla over the orbital border. Frequently a transmission by way of the veins is assumed and also a few casual suggestions occur in the literature that the processes may progress by way of the lymphatics. The latter view, however, has received very little attention.

In his chapter on the interrelationship of eye and teeth con-

ditions, Misch (1) makes the remark that a propagation by way of the lymphatics would always have to follow a roundabout course through the preauricular lymph glands. Hence, no certain case of a development in this way has yet been demonstrated. "It is true," he states, "that Birch-Hirschfeld (2) found a system of spaces in the orbital tissue, which is probably to be regarded as a lymph space system, but a communication of the lymph vessels of the orbit with those of the antrum of Highmore or the other accessory sinuses has so far not been demonstrated anatomically." The few hints in the literature, of a propagation along the lymph vessels may nevertheless be mentioned. In his report of orbital phlegmon Hirsch (3) describes four cases due to diseased teeth and expresses the view that in these cases the infecting agents from the infected peridental tissue were transmitted from the soft parts of the upper jaw to the orbit by way of the blood or lymph vessels. In further explaining, however, the process of propagation he only discusses a mode of transmission by the veins. Equally brief are remarks by Hillemanns (4) concerning the spreading of the process in his two cases of central retinitis arising from a diseased tooth to the root: "in both cases it was undoubtedly an inflammation extending from an inflamed tooth root to the posterior ocular pole by way of the lymph vessels or through the veins *per continuitatem*." No further comment upon the transmission by the former route is made by Hillemanns. Borel (5) simply states that the microbes and toxines from carious teeth are carried with the blood and lymph stream to the eyes. Mayrhofer (6) concludes from the anatomical study of his three cases that besides bony canals which have been described by Parinaud, the blood and lymph paths of the soft parts must be considered as probable routes of transmission. In his cases he found the fistulous duct embedded exclusively in the soft tissues. More positive is Pagenstecher's (7) statement that an inflammation of the orbital tissue may undoubtedly result from diseased teeth through direct infection, "which in all probability is transmitted through the lymph vessels."

Vossius (8) assumes in his case a rapidly ascending phlebitis, respectively lymphangitis. Gutmann (9) remarks concerning the pathogenesis of one of his reported cases of orbital phlegmon

due to infections from teeth: "Abscesses had developed in the orbit and the corresponding frontal sinus from the tooth alveolus by way of the lymphatics." He arrives at this conclusion from his microscopical findings. The route of transmission assumed in the other two cases is not the same. Generally such clear assertion that it was one or the other route is lacking. Not rarely the propagation by the vein is assumed, in spite of the distinct statement that indications of an actual phlebitis were not observed.

While these statements merely hint at the possibility that lymph vessels may carry infections from the teeth to the orbit, there is a positive reluctance on the part of authors to assign these channels any rôle in the extension of septic processes from the eye to the optic nerve and the meninges. This is particularly noticeable in Birsch-Hirschfeld who demonstrated a system of lymph spaces in the orbital tissue. This author as well as Merkel and Kallius (10) has also called attention to the interesting fact that the arteries are very loosely embedded in the orbital fatty tissue and that this is very probably due to the presence of perivascular lymph channels. Birsch-Hirschfeld makes the distinct statement that the distribution and the communications of lymph spaces which he found in this tissue are such as to provide definite channels for the propagation of inflammatory processes, and he adds: "Through the study of numerous microscopical preparations from a case of orbital phlegmon I have convinced myself that these spaces serve as routes for the invading leucocytes and lymphocytes." Yet he assumes a thrombophlebitis or a phlebitis or a periphlebitis in practically all cases where infections are transmitted from the orbit to the meninges and he even is an opponent to the belief of other authors that the processes may spread along the nerve sheaths. It would seem that it is not entirely consistent to admit a transmission of infections from the teeth to the eye by way of the lymphatics as well as a spreading of the processes in the orbit through the lymph spaces, while he interprets any further propagation from this region to the brain, without exception, as a phlebitis. It is only reasonable to assume that the lymph channels of the orbit as elsewhere in the body communicate with those of the neighboring regions and that lymph vessels accompany the blood vessels and nerves

through the orbital fissures and foramina and even the minute openings through the bone. Of interest in this connection are observations by Oeller (11), Leyden (12), and others of transmissions of infections along the nerve sheaths.

If we thoroughly study the anatomy and histology of the structures involved in these cases we will undoubtedly concede that the lymph vessels may play a primary and chief part. The veins may often be only indirectly the route of propagation. Gurwitsch (13) and others have demonstrated that the veins communicate with one another much more freely than the arteries and these communications are of special practical significance as Wetzel (14) has shown, for the reason that there are lymph spaces and lymph vessels in and about the walls of the veins, the course of which follows that of the veins. He calls attention to the fact from histology, that the tissue elements of the wall of the veins follow chiefly a longitudinal direction. This, of course, involves also an arrangement of the lymph spaces in a longitudinal direction. Inflammations and septic processes therefore progress preferably along the veins. They follow the lymph channels in the first instance; from the lymph spaces they spread in the adventitia, get access into the media, and finally invade the intima and thence spread in the rapidly forming thrombi. According to Benda (15) this course of phlebitis, in general, corresponds exactly to the picture of a localized phlegmon.

Mention must be made of an interesting observation made by Lartschneider (16 and 17) which refers to abscesses of the medullary spaces of the maxilla arising from diseased teeth and breaking through externally as fistula at the corner of the eye. The chief teeth concerned in these cases are the lateral incisors and the canines. Pus from diseased teeth may travel upwards under the periosteum of the anterior surface of the maxilla and reach the orbit *per continuitatem*, for the periosteum is directly continuous from the inner surface and border of the alveoli to the inferior and lateral walls of the orbit. Rupture of alveolar abscesses of the lateral incisor, labially and backwards, however, is not easy; they rather tend to break through upwards into the spongiosa and produce large abscesses of the bone, which Lartschneider has called "Markhöhlenabscesse," abscesses of the medullary spaces. They may remain latent for

years before breaking through externally. The diagnosis of such abscesses is difficult because neither swelling nor fluctuation is observed in them; but it is supported, according to Lartschneider, by the occurrence of swollen lymph tracts in the apical region of the involved lateral incisor at the plica of the mucosa. He warns, however, against mistaking mucus glands in the submucosa for enlarged lymph vessels. Of interest also are statements by Parinaud (18) concerning inflammations of the lacrimal sac in children. Contrary to the general view that inflammations from diseased teeth travel *per continuitatem* on the outer surface of the maxilla to the eye, he believes that pus from the infected tooth in his cases had traveled through canals into the substance of the jawbone and thence passed out into the skin through a fine opening in front of the lacrimal canal. The route which the pus takes in children in such cases is one provided for in the development of the teeth. That is to say, there are minute canals from the alveoli of the temporary teeth communicating with those of the second set of teeth and these latter are at a certain time so embedded in the anterior wall of the maxilla that the upper portion reaches the inferior orbital wall. Parinaud demonstrated the presence of these canals by injection of fluids.

Because these suggestions of a direct communication by lymph channels between the dental and the eye regions are, from the anatomical standpoint, quite vague and incidental, further investigations of this question seem indicated.

Demonstration of these communications by injection of the lymph vessels by the Gerota method such as has been successfully done with those of the dental pulp and the peridental tissue (19, 20, and 21) is almost impossible. The pressure must be considerable in order to force the injection fluid through the minute openings of the bone into the peridental tissue; the injection is followed by massage. If the pressure is not sufficient the fluid will not be driven far enough. I have, however, sometimes succeeded in filling deep and superficial lymph vessels of the infraorbital region of the face, simultaneously with those of the dental region. It is also obvious that injections in the region of the eye are unsuitable for such purposes; the injection mass will follow the direction of the least resistance and fill the loose tissue of the orbit. This occurs also in

injecting the fluid through the infraorbital foramen, which is the most accessible place to the eye as well as the dental region for reaching lymph channels in either direction.

In further experimental attempts to approach the question of anatomical relations between these two regions I followed the precedents of such writers as Orr (22), D'Abundo (23), and especially Homen (24). These investigators have shown that bacteria or their toxins experimentally or pathologically introduced into peripheral nerves are carried along the endoneural and perineural lymph channels to the spinal cord and brain and are capable of producing lesions in regions of the central nervous system quite remote from the seat of the original infection. With a similar point in view I have injected bacteria (staphylococcus, streptococcus, bacillus pyocyanus and haybacillus) into nerves of the face, into some of the lymph glands of the face, and into the subarachnoid space of rabbits. In some cases propagation of the bacteria from the place of injection, presumably along the nerve sheaths to remoter regions and occasionally even to the pulp of teeth, could be observed. However, the crucial point, to demonstrate beyond any doubt an exclusively lymphatic transmission by the presence of bacteria in genuine lymph channels lined with endothelial cells, is not easily settled to one's own satisfaction. Unless the bacteria are so numerous that they fill the lymph vessels to their capacity the delicate walls collapse.

Several of these animals were treated with vital staining fluids (trypan blue and lithium carmine) before being injected with bacteria. Observations in previous work on vital staining (25) led me to believe and express the view that the vitally stained granular cells in the connective tissue of all organs are intimately related to the lymph system. Possibly they are the endothelial cells lining the lymph channels at the very origin of the lymph system, having the property and function of absorbing the fluid from the tissue spaces and clefts as lymph proper. Lymph vessels which simply conduct the collected lymph to other lymph vessels, to lymph glands and venous outlets, are not lined with cells capable of taking the vital stain. With the extensive anastomoses existing in the lymph system it is possible to assume that bacteria injected into the tissue in the manner of the Gerota method might under favorable condi-

tions find their way into these primary lymph spaces and lymph capillaries whose true essential character as lymph channels is indicated by the vital staining of their endothelial cells.

I finally limited my efforts to an experimental study of the lymph supply of the eye only, making use of vital staining and of a drug by means of which Birch-Hirschfeld believes to have demonstrated lymph spaces and lymph vessels in the orbital tissue. This drug is paraphenylendiamine hydrochloride, the action of which is, according to this author, to cause dilatation of the capillaries and increase of the lymph excretion; and this is especially well demonstrated about the eye.

The results of my experiments are described in detail in a paper entitled, "A Contribution to the Study of the Lymphatic System of the Eye," which is being published in *The Anatomical Record*. The main observations may be briefly summarized here.

Experiments with vital staining, carried on for years, have led me to the conviction that the vitally staining cells which are found in the connective tissue of practically all organs of the body are intimately related to the lymph system. These cells have received various names, rhagiocrines, resting wandering cells, macrophages, pyrrol cells, histiogenic cells, etc., and their functional character has been variously interpreted; they are generally alleged to have secretory functions. The constancy with which these cells occur in the same tissues and organs, their systematic arrangement and distribution, their relation to the blood vessels, have led me to believe that they are the endothelial cells lining the minute lymph channels which constitute the beginning of the lymph system. The ability of the cells to take up the vital stain probably coincides with specific functional activities on their part, one of which may be to collect the modified plasma of the surrounding tissue as lymph proper. Lymph vessels which merely conduct the lymph have not vitally staining endothelial cells. The application of this presumed relationship of the vitally stained cells to the lymph system does not lead us to any conflict with anatomical data. Assuming that vitally stained cells in the connective tissue denote the presence of lymph channels we are struck by the constantly recurring observation that these cells

are only found where lymph vessels are known to be present or where they may be presumed to exist; they are invariably absent in those tissues which are known or suspected to be devoid of lymph vessels. I may mention here that Schnaudigl (26) who studied the effect of vital staining on the eye made observations concerning the occurrence of vitally stained cells which are on the whole in accordance with my own. He did not, however, draw any inferences as to their possible relationship to the lymph system.

The lens is devoid of vitally staining cells. No such cells are found in the cornea; but the conjunctival tissue overlying the corneal tissue shows such cells in large number. Occasionally a long slender vitally stained cell is seen to extend from the conjunctiva into the cornea. As to the sclera which contains such cells in scant number, it is not quite clear whether they belong to the tissue proper or whether they accompany the vessels traversing the scleral tissue. The cells are quite numerous in the scleral and corneal conjunctiva and are present in great abundance in the limbus conjunctivæ. The iris is very poorly provided with them; a cell is found here and there in the region towards the posterior chamber. This scarcity is the more striking as the organ, which it joins, is unusually rich in vitally staining cells, namely the ciliary body, and chiefly the ciliary processes. The cells are in proportion more plentiful here than in any other part of the eye. They are arranged along the blood vessels, from which, however, they are always at some distance. They are present in all processes, they are always in abundance and they are always arranged in the same way. When the injections of the staining fluid have been continued for some time these cells are very large and the granules are very coarse while in the iris or the sclera they remain small and slender, an observation to which also Schnaudigl has called the attention. This author believes that these cells have an almost dangerous affinity for the staining substance; they are very vulnerable and show the injurious effect of the dye after prolonged contact with the staining fluid. From the root of the ciliary processes the cells are observed to occur in smaller number all along the subjacent inner layer of the ciliary body; the deeper region facing the sclera, is scantily supplied with these cells and resembles the iris in this respect. In the choroid the

cells are found chiefly in the choriocapillaris. There are none in the retina. The endo- and perineural tissue always contains vitally stained cells. The loose orbital tissue is relatively poorly supplied with them. The muscles of the eye show the cells in the interfascicular and interfibrillar tissue, generally in the neighborhood of the blood vessels. They seem to be in closer relationship to the blood capillaries than is for example observed in the ciliary processes; they often seem to spin around the capillaries while these again wind about the muscle fibers. In the lacrimal gland they occur in the interacinous tissue and the connective tissue surrounding the glandular structures. The lids and the nictitating membrane are well supplied with them; they always occur in the same arrangement and the same distribution. The cartilage is absolutely free from such cells.

If we are to admit the view that these cells represent the endothelium of lymph capillaries and lymph spaces, we recognize that these findings correspond quite well with what we actually know or may reasonably presume concerning the lymph supply of the different parts of the eye.

Birch-Hirschfeld believes he has demonstrated a lymphatic apparatus of the orbit by means of paraphenylendiamine the effect of which in the dog and the rabbit is edema of the orbital tissue. I am not inclined to consider all the innumerable spaces which we find filled with fluid after diamine poisoning as dilated lymph spaces and lymph vessels. The fluid does not only fill all available spaces but also splits apart the fibers of muscles and the connective tissue. One of the chief effects of the drug seems to be on the blood vessels, perhaps irritating the endothelial wall of the capillaries and thereby rendering it more permeable. This is particularly noticeable in the muscles of the eye. Puppe (27) and Kunkel (28) also assign to the blood vessels a greater significance for the development of the intoxication edema.

On account of the extensive anastomoses of lymph vessels obstruction to the outflow of lymph does not readily occur. We may conceive of the action of paraphenylendiamine as such: in subcutaneous injections the drug enters the lymph channels; thence it is carried into the blood, where it irritates the walls of the blood vessels possibly having an injurious effect

on the endothelial cells of the capillaries. Transudation of the plasma occurs very rapidly, the fluid filling all the tissue spaces and separating the tissue elements. The function of the endothelial cells of the lymph capillaries being not simply one of absorption of such free fluid, but probably one of a selective action, of an interaction of metabolic processes, the fluid is not taken up and carried off rapidly enough with the result that stasis-edema develops. The value of paraphenylendiamine in conjunction with vital staining is in my opinion this, that it illustrates and supports the theory of a difference between spaces filled with tissue fluids and real lymph spaces and lymph capillaries, which constitute the channels of origin of the lymph system.

It has become customary to consider the ciliary body as the main source of the intraocular fluid. Hamburger (29) attributes also to the iris an important rôle in the function; in fact, he believes that every part of the eye participates in the secretion and resorption of the fluid. Wessely (30) is of the opinion that it comes nearest a transudate. It does not contain any substance which is foreign to blood serum. There is, hence, no reason why we should not consider the process of its secretion as a filtration process. Some difficulties arise from the relatively high salt content and the involved greater osmotic pressure, a property which it shares with the lymph. The most striking difference is no doubt the low albumen content which places it in one class with the cerebrospinal fluid, the anniotic fluid and the urine excreted in the glomeruli of the kidney, and on the other hand makes it stand in marked contrast to the lymph. Counterpressure to transudation may be the explanation; but we are quite as well justified in supposing that the presence of a special layer which covers the vessels may be the cause of the retention of the albumen. In the eye, the epithelium of the ciliary processes and the endothelium of the iris may act as such barriers.

Schnaudigl expresses the view that the vitally staining granular cells in the connective tissue of the ciliary bodies may be the chief agents in the secretion of the intraocular fluid. The epithelial cells covering the ciliary processes remain colorless in injections of trypan blue, an observation which I also made with lithium carmine. It does not seem permissible to

me to assume this specific function from the mere fact that these cells have a pronounced affinity for stains. For we must bear in mind that not only do vitally staining cells practically occur throughout the body in the connective tissue but also that invariably they are larger and more coarsely granular in definite regions of the body, for example in the pia of the brain where they occur in patches, in the choroid plexus, in certain regions of the nasal apparatus. On the other hand, there are cells admittedly endowed with secretory functions, cells other than those within the connective tissue which also have a pronounced affinity for vital stains (the epithelial cells of the choroid plexus, in the hypophysis, the thyroid gland, the syncytial cells of the placenta, the epithelial cells of the convoluted tubules of the kidney) while other cells of a similar type are not stained by trypan blue or carmine. I am more inclined to believe that inasmuch as these particular vitally staining cells occur practically everywhere in the connective tissue they have everywhere the same function to perform and inasmuch as in some localities they are invariably more intensely stained, they are involved either more intensely in the same process, or in another associated function. Along this line of reasoning we may also assume that the function of lymph secretion or lymph resorption is associated with the secretion of a fluid related to lymph such as the cerebrospinal fluid, the intraocular fluid or even the chyle. From this standpoint the great scarcity and smaller size of vitally staining cells in the iris would speak against the view of some writers that the iris is notably involved in the secretion of the aqueous humor while the presence of a large number of intensely staining granular cells in the ciliary body support the more generally accepted theory that these cells are the main source of the intraocular fluid. On the other hand there is nothing in my view of the part which the vitally staining cells play in the lymphatic apparatus that would contradict the view expressed by Hamburger that there is a more active resorption of the fluid by the iris through lymph channels than the generally assumed venous drainage into the canal of Schlemm. The fluid has a direct entrance into the iris through the crypts and thence into the lymph vessels. According to my observation lymph channels which simply convey lymph have no vitally staining endothe-

lial cells. This might explain the curious fact that there are so few of such cells in the iris, especially in the anterior portion. As to the drainage into the canal of Schlemm, Hamburger states that the resorption through the spaces of Fontana may also be along perivascular lymph spaces and not by the blood vessels.

Since indubitable evidences of an etiological relationship between infectious processes about the teeth and pathological conditions elsewhere in the body have been the indices for new therapeutical measures, any clinical and anatomical contribution must be considered as an aid or a stimulation to further investigative work. As such I offer these data from the literature and the mere suggestions of experimental observations along these lines. Affections of the eye from diseased teeth in their relations to the lymph system represent a field which still is practically unexplored.

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TENOTOMY OF THE INFERIOR OBLIQUE.[1]

By HARVEY J. HOWARD, M.A., M.D., Peking, China.

(With four photographs on Text-Plate XIV.)

THE diagnosing of motor anomalies of the eyes is receiving more serious attention on the part of ophthalmologists than ever before. It is now recognized that about one in every three or four eye patients that come to one's private office suffer from some defect in their eye movements. These patients generally come with a history of symptoms of asthenopia which they hope to relieve by the wearing of glasses. It is easy to overlook a pathologic muscle condition unless a fairly complete muscle examination is routinely made. When overlooked, it is quite possible that the glasses prescribed to correct ametropia actually aggravate the symptoms which in many cases are almost entirely due to a muscle imbalance.

Only a small proportion of muscle cases can be dismissed with simply the prescribing of glasses. Many of them are bound to tax the skill and patience of the ophthalmologist and require careful observation and various forms of treatment over more or less extended periods. Now and then a case is found, generally one of long standing, which is not amenable to treatment of any kind. Likewise the effect of corrective measures varies so greatly with different individuals that one must always give a guarded prognosis as to a complete cure. In many cases the patience of the patient is exhausted before the ophthalmologist exhausts his repertoire of treatment.

[1] Presented before the Eye Section of the China Medical Missionary Association at its Biennial Conference, Peking, China, February 21–28, 1920.

These cases obviously add nothing either to the good reputation or the comfort of mind of the doctor. It has been the experience of many men that once they tackled the study of eye muscles and eye movements in the same determined and intelligent way in which they tackled their primary medical education most of the mysteries of muscle cases cleared up, and the new enthusiasm which they had for such cases gave them a new hold on their patients.

To diagnose cases of simple latent lateral deviations is comparatively easy. A knowledge of the principles of muscle movements permits us, for example, to differentiate between a convergence insufficiency and a divergence excess; or between a primary convergence excess and a secondary divergence insufficiency on the one hand, and a primary divergence insufficiency and a secondary convergence excess on the other hand. It is when we are confronted with high degrees of hypertropia or hyperphoria that we find complete diagnosis more difficult. In long-standing cases especially of the acquired type we are apt to find that other motor anomalies have taken place, which have altered the original condition from a simple to a very complicated one. However, even in these cases, our task is made easier if we keep clearly in mind the normal physiological action of the eye muscles, both individually and collectively.

One of the most striking anomalies of an individual muscle is underaction of a superior rectus. This underaction is generally caused by some impairment of the nerve branch or the nerve nucleus supplying that muscle, in which case we speak of the deviation as paretic. Underaction may also be caused by an ill-development of the muscle itself, or by some fault in its origin or insertion, in which case the anomaly is called, according to Duane, a structural or an insertional deviation respectively. Underaction of a superior rectus muscle may be congenital or acquired. If congenital, the deviation is apt to remain noncomitant and for this reason gives as a rule the most typical picture. If acquired, the deviation always tends to become comitant, *i.e.*, by a gradual contraction of the direct antagonist, which in the case of the superior rectus would be the depressors of the same eye, the eye affected is made to stand lower than the other in all the fields of fixation. This enables the individual to more readily suppress the false image

which becomes less and less annoying as time goes by. Sometimes a head tilting is brought about, in order either to compensate for the lower position and consequently the higher image of one eye, or to more readily effect a suppression of the false image.

Recently a number of these cases have been published as having been treated with either total or partial relief of symptoms by tenotomy of the inferior oblique of the opposite eye. This muscle is the associated antagonist of the superior rectus of the other eye, which means that these muscles as a pair perform a coördinated or an associated movement of the two eyes —a movement evidently controlled by a common center in the brain. So comparatively few cases of tenotomy of the inferior oblique have been published with full reports that there is only a small amount of data on hand as to what degree of correction of a deviation in a vertical plane we may expect from such tenotomy. It is therefore for the purpose of making a small contribution to this subject that I desire to report somewhat in full two cases which recently came under my care and observation.

CASE I.—S. C., age 9, son of a missionary. History of "crossed eye" for several years. Father thinks boy may have had it since birth, but not sure. Seems stupid in his school work.

Vision without correction: R. = $\frac{20}{30}$ − 2; L. = $\frac{20}{30}$ − 3.

Vision with the following correction which has been worn for the past three or four years: R. + 1.00 sphere, + 0.75 cyl. @ 90° = $\frac{20}{30}$ + 2; L. + 1.25 sphere, + 0.75 cyl. @ 90° = $\frac{20}{30}$ + 2.

A. Tests without glasses showed:

I. With screen and parallax.

1. Esotropia for distance of 12$^{\Delta}$ and for near of 16$^{\Delta}$. The esotropia was generally manifested by the left eye although fixation with the left eye could be maintained for a brief period (Fig. 1).

2. Right hypertropia with the left eye fixing of 30$^{\Delta}$ and a left hypotropia with the right eye fixing of 20$^{\Delta}$.

3. A 25$^{\Delta}$ prism vertically placed before one eye and a red glass before the other brought the two images on a horizontal line. There was, therefore, an anatropia of 5$^{\Delta}$, for the 25$^{\Delta}$ prism undercorrected the right hypertropia by 5$^{\Delta}$ and overcorrected the left hypotropia by the same amount. This phenomenon was demonstrated by the screen and

parallax test first before one eye and then the other while the prism was in position, when it was found that the eyes came down an amount equal to 5$^{\Delta}$ in each case when the screen was removed.

II. Using a straight line on a card rather than a small object like a pin's head, the relative near point of convergence was found to be 75*mm* which was calculated from the center of rotation of the eyeball. The convergence angle was 39°.

III. Testing the associated movements of the two eyes showed:

1. Marked underaction of the left superior rectus and overaction of the right inferior oblique, both elicited by the position of the eyes when the patient looked up and to the left (Fig. 2).

2. Underaction of the right inferior rectus and overaction of the left superior oblique, both elicited by the position of the eyes when the patient looked down and to the right.

3. Other associated movements normal.

IV. With the Worth-Black Amblyoscope the extent of the eye movements was recorded as follows:

Eyes to the right.....	right eye	50°,	left eye	45°.
Eyes to the left.....	" "	52°,	" "	45°.
Eyes up and to the right..	" "	37°,	" "	35°.
Eyes down and to the right	" "	23°,	" "	40°.
Eyes up and to the left	" "	40°,	" "	5°.
Eyes down and to the left	" "	30°,	" "	25°.
Eyes up.....	" "	40°,	" "	30°.
Eyes down.....	" "	40°,	" "	40°.

These figures confirm very conclusively the results of the tests for the associated movements as given above. It was also proven that simultaneous vision with the two eyes existed, but not binocular fusion when the hypertropia was corrected by the proper adjustment with the amblyoscope.

V. Skeletal measurements showed, when patient stood fixating an object directly in front:

1. A head tilting to the left which made a difference of 7*mm* in level between the right and left eyes.

2. Left shoulder 7*mm* higher than the right. When head was forcibly straightened, the elevation of the left shoulder increased indicating a certain amount of torticollis.

3. The spine showed a slight double lateral curvature in the thoracic region, the upper curve being to the right and the lower to the left.

VI. Refraction following atropine cycloplegia showed:

ILLUSTRATING DR. HARVEY J. HOWARD'S ARTICLE ON "TENOTOMY OF THE INFERIOR OBLIQUE."

FIG. 1.—Right eye fixing, left hypoesotropia, left head tilting.

FIG. 2.—Looking up and to the left. Marked underaction of left superior rectus and spasm of right inferior oblique.

FIG. 3.—Right eye fixing with full correction on. Left hypoexotropia. Head was set straight by photographer.

FIG. 4.—Left eye fixing with full correction on. Right hyperexotropia.

R. + 4.25 sphere, + 0.50 cyl. @ 90° = $\frac{20}{20}$ + ; L. + 4.00 sphere, + 0.50 cyl. @ 90° = $\frac{20}{20}$.

B. Tests with full correction on showed:

Practically the same results were obtained as were found before without correction with one striking exception. There was an alternating exotropia of about 36Δ for distance as against an esotropia of 12Δ without glasses, which made a difference of 48Δ in the lateral deviation (Figs. 3 and 4).

The full correction was prescribed and after six months another examination was made which confirmed the earlier findings.

Inasmuch as the chief defect seemed to be that of the left superior rectus, it seemed advisable to tenotomize the inferior oblique of the right eye for the purpose of putting the visual axes of the two eyes more nearly into the same plane. It was not anticipated that such an operation would produce more than a partial correction. Not being associated with a hospital in New York City at that time, I asked my friend Dr. J. W. White, to whom I am indebted for the photographs of the case, to do the tenotomy, which he did. He "found an anomalous attachment of this muscle to the orbital plate of the superior maxillary bone. Instead of one insertion, there were two, one of which was found far to the outer side."

Five weeks later, I made another examination of the boy with the following result:

Without glasses: Esotropia for distance of 2Δ. Right hypertropia (with left eye fixing) of 30Δ. Left hypotropia (with right eye fixing) of 10Δ.

With full correction: Exotropia for distance of 16Δ. Right hypertropia of 30Δ. Left hypotropia of 10Δ.

With a 20Δ prism vertically placed before one eye and a red glass before the other, two images were still visible, but they were on a horizontal line. By using the screen and parallax test, first before one eye and then the other when the prism was in this position, it was found that each eye came down behind the screen an amount equal to 10Δ, *i.e.*, there was an anatropia of 10Δ.

The operation, therefore, reduced the average deviation in a vertical plane by 5Δ, but it increased the anatropia by the same amount. The strange thing was that there was no decrease in the hypertropia of the right eye which was naturally expected from a cutting of one of its elevators. The decrease, however, in the hypotropia of the left eye must be attributed to the operation. That the effect occurred apparently upon the left eye rather than upon the right can be explained by the possibility of the patient tilting his head backwards a little more than he did before the operation, which would tend to neutralize somewhat the spastic action

of the left superior oblique. That the same measurements of the vertical deviation were obtained by testing again and again would justify the conclusion that the position of the head in the primary position was permanently altered by the operation. It should be stated that the operation did not appreciably alter the head tilting to the left.

The most marked changes occurred in the lateral deviations. Without glasses the esotropia was reduced from 12Δ to 2Δ, *i.e.*, a difference of 10Δ. With the full correction on the exotropia was reduced from 36Δ to 16Δ, or a difference of 20Δ. In other words the difference in the lateral deviation without glasses and then with glasses was 18Δ as against the 48Δ that existed prior to the operation. If 18Δ represents approximately the effect of relaxation of accommodation that occurred after the operation when the full correction was put on, then the balance of 30Δ in the lateral deviation must be attributed in some way to the operation. In this connection it should be remembered that the inferior oblique muscle is an abductor as well as an elevator and that this power of abduction increases as the eye turns out, in the same proportion as its power as an elevator increases as the eye turns in. Therefore 20Δ of this 30Δ (or the difference between the exotropia of 36Δ before the operation and of 16Δ after operation obtained with glasses on) can be attributed to cutting of the inferior oblique of the right eye. The remaining 10Δ of this 30Δ is not as easily explained. Theoretically there should either have been no difference or possibly an actual increase in the esophoria without glasses before and after the operation, because with one abductor cut the adductors would tend to proportionately overact. True muscle conditions, however, are elicited only when any other contributing factor is eliminated. Therefore, in making a diagnosis we must primarily consider the findings when the eyes were made emmetropic.

From the father's statements as to the length of time the boy's eyes were affected, from the absence of history of injury or severe infectious disease, there is every reason to consider the condition congenital in origin. An exploratory operation was not done to determine whether there was an absence of the left superior rectus or a faulty insertion. Neither can we state that there existed any impairment of the nerve or nerve-nucleus supplying that muscle. The evidence indicating the possibility of an insertional anomaly was the fact that there was found an anomalous attachment of the inferior oblique of the right eye; also the marked correction of the exotropia following operation. The evidence, however, is not sufficient to warrant a more specific diagnosis than that of congenital underaction of the left superior rec-

tus. The overaction of its associated antagonist, the right inferior oblique, could, however, easily be a secondary condition. An inability to look up and to the left although the necessity to do so is frequent and the desire is strong, naturally produces an overexcitation of the brain center controlling that pair of muscles, which while producing no response in the superior rectus, ultimately produces a spasm of the inferior oblique.

In view of all the above facts, the following *diagnosis* was made:

Congenital underaction of the left superior rectus and right inferior rectus; overaction (probably secondary spasm) of the right inferior oblique and left superior oblique; alternating exotropia with right hypertropia and an anatropia.

CASE 2.—Mrs. M. M. W., age 46, American. History of severe fall eighteen months before. Struck head on the left side at about the junction of the temporal, parietal, and occipital bones. Unconscious for several hours. Followed for six weeks by amnesia which gradually cleared up. Eye symptoms consisting of dizziness and diplopia for part of the time began immediately following consciousness. Menses at the time of the injury were occurring every two or three months. Following the injury they completely stopped and never returned. In view of the age of the patient this cannot be considered other than a coincidence.

Results of examination: Vision R. = $\frac{20}{30}$; L. = $\frac{20}{30}$.

Following homatropine refraction: Vision, R. + 0.50 cyl. @ 105° = $\frac{20}{20}$; L. + 0.50 cyl. @ 120° = $\frac{20}{20}$.

Screen and parallax test showed: Exophoria for distance of 5Δ. Exophoria for near of 15Δ. Right hyperphoria of 8Δ. Prism sursumvergence power of 8Δ.

With red glass before one eye, diplopia was always immediately induced showing that the patient was maintaining fusion only with great difficulty.

Near point of convergence = 160*mm.*

Associated movements' tests showed nearly complete underaction of the left superior rectus and some overaction of the right inferior oblique.

Diagnosis.—Traumatic paresis of left superior rectus and secondary spasm of right inferior oblique; primary convergence insufficiency with secondary divergence excess.

The condition seemed to call for a tenotomy of the inferior oblique of the right eye, which I did using novocaine injection.

At the end of a week, examination showed only 1Δ of right hyperphoria remaining. The exophoria was unchanged, viz. 5Δ for distance and 15Δ for near. At the end of three

months another examination showed the right hyperphoria still to measure 1Δ. The exophoria had been reduced slightly, *i.e.*, to 3Δ for distance and to 13Δ for near. This reduction may have been brought about because the patient was resuming more and more the use of her eyes for near work, which she had totally avoided during the interim of the injury and the operation. This exercise would tend to increase her convergence power and in her case reduce the exophoria, although the latter result does not generally nor necessarily follow.

The patient was not subsequently seen, but at the last examination mentioned above, her symptoms of dizziness and frequent diplopia had absolutely disappeared. She was so much improved mentally and physically that she seemed like a different woman. Her only complaint was that her eyes quickly tired while doing near work. This was aggravated by the complication of a beginning presbyopia which called for a small plus correction in addition to the cylinders she wore, but which could not be given until her exophoria for near was considerably reduced. For this purpose she was instructed how to perform converging exercises for a period of several months and then report for another examination.

The operation itself produced an immediate and a permanent reduction of 7Δ in her hyperphoria in the primary position. The 1Δ of hyperphoria remaining was easily within her prism sursumvergence power which at the time of the operation was as much as 8Δ. Her ability to look up and to the left was of course considerably limited, but there was no diplopia whenever she did so, for the reason that the underaction of the pair of associated antagonists which functioned in that field was equal and symmetrical.

The effect of the tenotomy in the first case was a reduction of 5Δ in the vertical deviation. From the standpoint of the patient the effect was merely a cosmetic improvement largely due to a reduction in the exotropia. The effect of the tenotomy in the second case was a reduction of 7Δ in the hyperphoria, which from the patient's standpoint resulted in a complete cure. Judging from the results of these cases and of other cases published, I suggest for vertical deviations of 10Δ and over, due primarily to an underaction of a superior rectus, that an exsection of the inferior oblique of the opposite eye be performed rather than a simple tenotomy. Such a procedure can do no harm and it is quite possible that by it a greater permanent correction can be obtained.

It seems justifiable also to suggest the probability that anatropia is a far more common condition than is generally supposed. Whenever there is found a difference in the amount of the hypertropia and the hypotropia, there theoretically should and probably does exist an anatropia or a catatropia which is equal to one half of this difference. We should therefore, particularly in all cases of large deviations in a vertical plane, not only ascertain the degree of elevation of one eye, but also the degree of depression of the other.

OBSERVATIONS ON ONE HUNDRED CASES OF PRIMARY (ACUTE CONGESTIVE AND SUBACUTE) GLAUCOMA UNDER TREATMENT AT THE ROYAL LONDON OPHTHALMIC (MOORFIELDS) HOSPITAL DURING THE PAST FOUR YEARS WITH A COMPARISON OF VISUAL RESULTS.[1]

By CHARLES MAGHY, M.D., Chicago, Ills.

Late Pathologist to Royal London Ophthalmic (Moorfields) Hospital.

IF ophthalmologists knew what maintained the normal intraocular pressure, they would be better able to understand the pathological cases, but until such time has arrived, much must remain unexplained in this branch of ophthalmology.

The classification followed in this series of cases was the one suggested by Colonel Elliot, namely, acute congestive and non-congestive glaucoma. Many of the acute cases, however, showed that a condition of chronic glaucoma had existed in the other eye at some period. No attempt was made to divide the cases into the various stages. Aside from operations, the treatment was the same for each case with the exception that most of the severe cases had leeches applied. Oily eserine 1% was used two or three times a day during the first twenty-four hours, followed by the aqueous solution ½% three times a day. Aspirin was occasionally given in large doses but morphine was seldom resorted to for the pain. The operated cases received calomel and salts, except those taken direct to the operating-room from the out-patient department. In all the trephined cases the Lang 1½*mm* instrument was used, after being tested

[1] Read before the Chicago Ophthalmological Society, Oct., 1919.

with fine kid and examined with a binocular loupe. Most cases had one stitch of fine black silk in the conjunctival flap. A few patients had a Herbert's sclerotomy, but most of the acute cases had an iridectomy, the iris being torn out after being severed at one edge. A narrow Graefe knife was used to make the section.

Following the operation, atropine sulphate ¼% was instilled in the operated eye by some surgeons, in other cases it was not used until the second day, when the anterior chamber was usually well reformed. Only the operated eye was bandaged.

The tension was seldom recorded with a Schiotz or McLean tenometer prior to operation. Fields were taken in nearly all the cases where the vision would permit, but chiefly at a later period when the eye had become quiet, following the operation for the relief of tension. Many of the cases did not attend the hospital for a long period after being discharged, so that it was impossible to keep a record of their progress.

Eliza W., aged 57, married, general health good. Admitted November 9, 1918, under Mr. Lawford. Patient contracted influenza October 18th. Left eye showed a condition of acute congestive glaucoma. Vision equaled hand movements with projection accurate. Greenish reflex. Fundus obscured. T. + 2.

November 13, 1918, left iridectomy under $CHCl_3$.

Discharged, November 29th. Left eye was quiet. Vision with −1.00 sph. and −4.00 cyl. was $\frac{6}{12}$. Lens uninjured. O. D. was not cupped, color was good. Tn.

Right eye. Vision with a + 3.00 sph. equaled $\frac{6}{6}$. O. D. normal. Tn.

Emma H., aged 57, married, general health good. Admitted, December 12, 1918, under Mr. Lawford. December 2, 1918, patient was struck in left eye by a piece of wood. Treated by a chemist with lotion but no "drops." On admission left eye showed a condition of acute congestive glaucoma. The cornea was not abraded and did not stain. No keratitis punctata or synechiæ were seen. Fundus was obscured. T. + 1. Vision equaled hand movements. December 15th nasal field contracted about five degrees between fifteenth and seventy-fifth meridians. A 10*mm* white square test object was used and the day light.

December 17th was discharged. Left eye was quiet.

Cornea was bright. Anterior chamber was shallow. Vision with a + 2.00 sphere equaled $\frac{6}{6}$. Tn. O. D. normal.

Right eye. Vision $\frac{6}{6}$; vision with a + 2.50 sph. was $\frac{6}{6}$. Anterior chamber was shallow. O. D. was normal. Tn.

Hannah H., aged 50, married, general health good. Had pyorrhœa very badly. Admitted July 13, 1918, under Mr. Lawford. Patient contracted influenza the last week in June. The left pupil was dilated, oval, and fixed. The optic disk was not cupped. Vision equaled $\frac{6}{36}$. The nasal field showed a contraction of 10 degrees between the fifteenth and ninetieth meridians. T. was + 1. The right eye was perfectly quiet.

On July 16th, the right eye suddenly developed an attack of acute congestive glaucoma (no eserine had been used in this eye, as the tension was normal on admission) and on the 17th, an iridectomy was necessary. The right vision, taken repeatedly in August, remained $\frac{6}{18}$ while on admission it was $\frac{6}{6}$ with correction.

Although the left eye was apparently normal, the tension remained + in spite of treatment, and on August 10th, an iridectomy was done under $CHCl_3$. On discharge, August 24th, the right vision with a + 3.00 sph. and − 6.00 cyl. was $\frac{6}{6}$. L. V. with a + 4.50 sph. and a − 6.00 cyl. was $\frac{6}{6}$. In neither case was the optic disk cupped. Tn. in each when discharged.

Harvey K., aged 70, married, general health good. Admitted May 29, 1918, under Mr. Lawford. On admission right vision was $\frac{6}{60}$. Fundus obscured. T. + 2. The patient had no treatment prior to admission.

June 1st, right eye was trephined under cocaine. June 11th, the vision was $\frac{6}{24}$. O. D was not cupped. Quiet. Tn. The left eye had occasional attacks of obscured vision the past eighteen months. On admission the eye was perfectly quiet. Vision equaled hand movements. T. was + 2. The optic disk was cupped. June 14th, left eye trephined—cocaine.

Discharged, June 22d. R. V. was $\frac{6}{18}$. L. V. was hand movements. Patient never had acute pain in his left eye.

Ellen S., aged 68, married; subject to rheumatism. Admitted May 3, 1918, under Mr. Lawford. No treatment. On admission, the right eye showed a condition of acute congestive glaucoma. The vision was reduced to counting fingers. T. was + 1. May 4th, had an iridectomy on right eye under $CHCl_3$.

Discharged, May 25th; right eye was quiet. Vision was $\frac{6}{24}$. Pillars were free. O. D. was not cupped. T. was −.
Left eye. Quiet. Full field. O. D. was normal. Vision equaled $\frac{6}{9}$. Tn.

Charlotte T., aged 61, widow, good health. Admitted January 11, 1918, under Mr. Lawford. Her present attack of pain in the right eye began in the early morning of January 3d. On admission the right eye showed a condition of acute congestive glaucoma. Vision was $\frac{6}{60}$. T. + 2. January 12th had a right iridectomy under $CHCl_3$.
Discharged, February 6th. Right eye was quiet. Vision with a + 2.50 cyl. was $\frac{6}{18}$. Iris pillars were free. Lens uninjured. O. D. was not cupped. Tn.
Left eye. In June, 1902, had an iridectomy at Moorfields Hospital for chronic glaucoma. Vision equaled hand movements. O. D. was cupped and atrophied.

Maud S., aged 31, single, good health. Admitted to Moorfields Hospital January 21, 1918, under Mr. Fisher. On January 19th, patient had her first attack of pain in each eye between midnight and dawn. Her family physician gave her morphia for the pain and vomiting. No eye treatment was instituted. January 21st, the day of admission, each eye showed a condition of acute congestive glaucoma. The vision was reduced to counting fingers. Right T. was + 2. L. T. was + 1. Treated with eserine forty-eight hours. January 23d had an iridectomy on each eye under CHCl3.
Discharged February 13th. Right eye showed nothing of special note beyond a small, rounded hemorrhage, about two disk diameters below the disk. Vision equaled $\frac{6}{12}$. Pillars were free and the lens was uninjured. Tn. O. D. was not cupped. Left eye was quiet. Vision was $\frac{6}{18}$. O. D. was not cupped. Iris pillars were free. Lens was uninjured and the tension was normal.

Issac P., aged 41, married, good health. Admitted July 17, 1918, under Mr. Fisher. On July 5th, patient had his first attack of pain in the right eye. No treatment prior to admission. T. was plus and the eye was painful. Vision equaled hand movements. Fundus obscured. July 22d, right eye was trephined under novocaine. The next day the anterior chamber was almost full of blood. There was no history or signs of an injury to the eye.
Discharged August 8th. The right eye was slightly injected. A small hyphæma was present. Vision equaled P. L. with projection accurate. Pupil remained dilated although this case received no mydriatic at any time. Fundus ob-

scured by vitreous haze. Tn. Trephine opening was apparently working well. Tn. Left eye—normal. Vision equaled $\frac{6}{6}$.

Grace B., aged 25, single, domestic, general health good. Admitted January 10, 1918, under Mr. Fisher. She had no treatment. The pupil was semi-dilated and acted fairly well. The margins of the optic disk were slightly blurred. No cupping was present. Vision was $\frac{6}{12}$. Tension with the Schiotz tenometer registered 38*mm Hg*. Arterial pulsation of the retinal vessels was present. January 11th, 14th, and 18th, patient had a right paracentesis under cocaine. In spite of this and eserine, the tension remained pathological, and on January 25th a Herbert's sclerotomy was performed.

Discharged February 1st. Cornea was bright. Pupil was active and circular. Vision equaled $\frac{6}{6}$. O. D. was slightly blurred. Tn.

Left eye. Appeared normal. Vision equaled $\frac{6}{6}$. Tension with Schiotz registered 19*mm Hg*. Optic disk was normal.

Harriet W., aged 61, married, good health. Admitted February 1, 1918, under Mr. Fisher. On January 28, 1918, the right eye was slightly painful for the first time. She was treated by her family physician with "drops." On admission the right eye showed a typical attack of acute congestive glaucoma. There were about 20 degrees of convergence present. Vision equaled counting fingers. Faint red reflex. T. + 1. February 1st, had a right iridectomy under novocaine.

Discharged February 15th. Cornea was bright. Iris pillars were free. Anterior chamber was shallow. Vision equaled $\frac{6}{60}$ unaided and was not improved. Optic disk was normal.

Left eye. Operated at Moorfields January 14, 1908, for acute glaucoma. Vision equaled $\frac{6}{12}$. O. D. was cupped. Iris pillars were free. Tn.

Emily H., aged 63, married, subject to rheumatism. Admitted May 2, 1918, under Mr. Hudson. Right eye became painful the first week of April. No treatment prior to admission. On admission right eye showed a condition of acute congestive glaucoma. Vision equaled counting fingers. Faint red reflex. T. + 1, with the Schiotz tenometer, 42*mm Hg*. May 2, 1918, right eye trephined under novocaine. May 6th, a small hemorrhage was seen two disk diameters to nasal side of O. D.

May 23d, discharged. Right eye was quiet. Anterior chamber was reformed but shallow. Cornea was bright.

No keratitis punctata seen. Vision was $\frac{8}{60}$. Vitreous opacities. Tn. O. D. was not cupped.

Left eye. Vision was $\frac{6}{6}$. Anterior chamber was shallow. Normal disk. Tn.

Edith N., aged 53, married, good health. Admitted June 14, 1918, under Mr. Collins. Treated by her family physician with "drops" prior to coming to Moorfields Hospital. Fundus was obscured. R. Vision equaled hand movements. T. with Schiotz tenometer was 40*mm Hg*. June 14, 1918, she had an iridectomy under $CHCl_3$. The incision was made with a keratome.

July 4th, discharged. The right eye was quiet. The cornea was clear. A small hyphæma was present. Iris pillars were free. The vitreous was hazy. O. D. was cupped. Vision equaled $\frac{4}{60}$. Tn.

Left eye. Quiet. Anterior chamber rather deep. Pupil was active and circular. Normal disk. T. with Schiotz tonometer was 30*mm Hg*. Vision equaled $\frac{6}{6}$.

Louisa T., aged 62, single, good health. Admitted February 6, 1918, under Mr. Greeves. L. Vision equaled shadows, projection was accurate. T. with Schiotz tenometer was 39*mm Hg*. Fundus obscured. Present attack was of seventy-two hours' duration. February 6th. L. glaucoma, iridectomy under novocaine.

Discharged February 27th. Cornea was still hazy. Iris pillars were free. Many vitreous opacities obscured the optic disk and fundus details. Vision equaled $\frac{6}{60}$. May 15, 1918, L. V. $\frac{6}{12}$.

Right eye. Operated for chronic glaucoma (iridectomy), September 5, 1901. Vision equaled $\frac{6}{60}$. Optic disk was cupped.

Rachel S., aged 48, married, good health. Admitted, February 4, 1918, under Mr. Greeves. The only treatment patient had was hot applications at home. On admission the eye showed a state of acute congestive glaucoma. L. Vision equaled $\frac{4}{60}$. Fundus was obscured. Tension with Schiotz registered 40*mm Hg*. February 6th, left eye was trephined.

February 16th, discharged. Left eye was quiet. Cornea was bright. O. D. was slightly cupped. Vision equaled $\frac{6}{12}$. Tn.

Right eye. Quiet. Vision was $\frac{6}{12}$ unaided. O. D. normal. Tn.

Annie C., aged 75, married, general health fairly good. Had rheumatism about twenty years ago in her joints.

Admitted December 10, 1917, under Mr. Parsons. Treated by her family physician. On admission the right eye showed an attack of acute congestive glaucoma. December 10th R. iridectomy under $CHCl_3$. December 11th, when I dressed the wound, the lens was between the lids. The lens capsule was opened when section was made. The eye, however, showed nothing of special note. The vision on admission equaled hand movements. T. was + 2.

January 5th discharged. R. quiet. No keratitis punctata but the iris pillars were both drawn up to the wound. Some lens material was present in the coloboma area. Vision equaled hand movements with projection accurate. Tn.

Left eye was quiet. Lens opacities. Vision was $\frac{6}{18}$. Field showed a concentric contraction of approximately 10 degrees. T. was normal.

Harriet H., aged 65, single, good health. Admitted, April 29, 1918. Left eye was first inflamed and painful April 15, 1918. Treated by her family physician with "drops and lotion." On admission eye showed a state of acute congestive glaucoma. Vision was reduced to hand movements. Fundus obscured. T. + 1. May 4th had a paracentesis under cocaine. Treated with eserine until May 14th without much success. L. iridectomy under $CHCl_3$ May 14th. The next day a large hyphæma was present, although there was no history of injury to the eye.

May 31st discharged. Left eye was quiet. Cornea was bright. Iris pillars were free. The anterior chamber contained some blood. Vision equaled $\frac{6}{6}$—due to hyphæma. O. D. was not cupped. T. was normal.

Right eye was blind from chronic glaucoma. Vision equaled P. L. T. was + 1. Lens opacities. Faint reflex.

Rose O., aged 48, widow, good health. Admitted March 21, 1918, under Mr. Spicer. On admission left eye showed a condition of acute congestive glaucoma. Vision was $\frac{6}{36}$. Fundus details were obscured and the T. was + 1. The left field showed a nasal contraction of less than 10 degrees between the 30th and 120th meridians. May 22, 1918, an iridectomy was done under $CHCl_3$.

April 2d, discharged. Left eye was quiet. Cornea was clear. Iris pillars were free. Vision was $\frac{6}{18}$. O. D. was not cupped. Tn.

Right eye was quiet. Vision equaled $\frac{6}{6}$. Normal fundus. Tn. Full field. June 25, 1918, R. V. was $\frac{6}{6}$. L. V. $\frac{6}{6}$.

Emma H., aged 56, married, good health. Admitted, November 22, 1917, under Mr. Hepburn. Used "drops" from a

general practitioner, until the 22d. On admission the right eye showed a condition of acute congestive glaucoma. Vision was hand movements. Faint fundus reflex. T. was + 2. November 23, 1917, an iridectomy was done under $CHCl_3$.

December 6th, discharged. Right eye was quiet. Vision was $\frac{6}{60}$ (lens opacities). Iris pillars were free. Vitreous opacities. O. D. was not cupped and the color was good. Tn. Left eye was quiet. Vision equaled fingers. Lens opacities. T. was normal. Many floating opacities were seen in the vitreous. Patient has since had a combined extraction but the vision was only $\frac{6}{60}$ in June, 1918, due to the vitreous opacities chiefly. The macula was normal.

Harriet M., aged 57, married, good health. Admitted May 28, 1918, under Mr. Hepburn. On admission the right eye showed a state of acute congestive glaucoma. Vision equaled fingers. T. + 1. May 30th right trephined under cocaine.

June 20th, discharged. Right eye was quiet. Vision was $\frac{6}{60}$. Tn. O. D. was cupped and pale.

Left eye. Quiet. Was an in-patient at Moorfields in August, 1912, with a hypopyon ulcer. Vision only light perception. Leucoma corneæ. T. +.

Rebecca B., aged 71, single, very deaf, good health. Admitted, March 25, 1918, under Mr. Hepburn. Treated by her doctor with "drops." L. Had acute congestive glaucoma on admission. Vision was hand movements. T. + 1. March 26th had left iridectomy under $CHCl_3$.

The right eye on admission was quiet. Vision was $\frac{6}{24}$. Vision with a − 3.00 sph. was $\frac{6}{6}$. Tn. Fundus normal.

About six hours after the left eye was operated, the right eye developed an attack of acute congestive glaucoma. The vision was only hand movements. T. + 1. The right eye was treated until March 28th and then had an iridectomy under $CHCl_3$.

On discharge, April 18, 1918, both eyes showed a very similar condition, the vision in each being $\frac{6}{18}$ unaided. Tn. Iris pillars were free. The disks were not cupped.

Sarah W., aged 64, married, good health. Admitted September 12, 1918, under Mr. Hudson. No treatment. On admission right eye had acute congestive glaucoma. Vision equaled P. L. T. + 2. September 12th an iridectomy was done under $CHCl_3$; incision entirely corneal.

Discharged, September 26th. R. V. equaled P. L. Cornea was slightly hazy. Iris pillars were free. Vitreous opacities. O. D. was obscured. Tn.

Left eye. Quiet. Vision was $\frac{6}{12}$. Nasal field showed about 5 degrees' contraction, chiefly above. O. D. slightly cupped, color was good. Tn.

Mary C., aged 82, married. Admitted, January 3, 1918, under Mr. Fisher. December 26, 1917, left vision failed rather suddenly after the eye was struck by an elastic band. Treated by her family physician with "drops" prior to admission to Moorfields. Left eye had acute congestive glaucoma, on admission. Vision equaled hand movements. T. + 2. Fundus was obscured. Treatment failed to reduce the tension to normal. January 5th left iridectomy under $CHCl_3$.

Discharged, January 21st. Left cornea was still slightly hazy. Anterior chamber was well re-formed. Iris pillars were free. Lens was clear. Vitreous opacities obscured the fundus details. Vision equaled fingers. T. was normal.

Right eye was excised at Great Northern Hospital in 1906.

George G., aged 57, married, soldier, good health. Admitted, January 23, 1918, under Mr. Collins with an attack of acute congestive glaucoma in the left eye. Vision equaled fingers. Fundus obscured. T. + 1. January 24th left trephined under novocaine.

February 11th discharged. Left was quiet. Vision was $\frac{6}{18}$. O. D. was normal. Tn. Drainage was perfect.

Right eye. Sight failed in 1913. Glaucoma iridectomy same year. History was that of chronic glaucoma.

William C., aged 67, widower, good health. Admitted March 9, 1918, under Mr. Hepburn, with acute congestive glaucoma, left eye. Vision equaled $\frac{2}{60}$. Fundus was obscured. T. + 1. March 10th an iridectomy was made under $CHCl_3$.

Discharged, March 20th. Cornea was bright. Pillars were free. Vision with a + 2.50 sph. was $\frac{6}{12}$. O. D. was normal. Tn. Right eye was operated at Moorfields Hospital for acute congestive glaucoma on April 11, 1916. Vision equaled $\frac{6}{60}$. Macular changes. Tn.

Mathilda O., aged 46, single, dressmaker, good health. Admitted February 2, 1918, under Mr. Hepburn. Right eye became inflamed and painful January 30, 1918. Was treated with eserine and hot bathing by her family physician. On admission vision was $\frac{6}{9}$ unaided. Full field. T. + O. D. was not cupped. February 5th an iridectomy was done under $CHCl_3$.

Left eye. Patient saw halos at times the past two years.

Never had emesis or severe pain. Vision was $\frac{6}{6}$. O. D. was normal. Tn.

February 21st, discharged. Right and left vision was $\frac{6}{6}$. Right pillars free. Tn.

Emma M., aged 42, married, good health. Admitted July 4, 1918, under Mr. Worth. On admission the right eye showed a state of acute congestive glaucoma. Treated with eserine, heat, and leeches. Vision on admission was hand movements.

Left eye. Operated for chronic glaucoma at St. Thomas's Hospital, 1912. Vision equaled shadows. O. D. was pale and cupped. Fundus showed old choroidal changes.

July 15, 1918, discharged. Right eye was quiet. Cornea was bright. O. D. was not cupped. Vision equaled $\frac{6}{60}$. Fundus showed old choroidal changes. Tn.

Charlotte M., aged 67, married, good health. Admitted May 17, 1918, under Mr. Flemming. February 5th patient had her first attack of obscured right vision. Came as an out-patient March 12th. R. V. was $\frac{6}{18}$. Vision with a $-$ 2.00 sph. was $\frac{6}{6}$. May 17th, homatropine and cocaine were put in each eye by mistake by the nurse in the out-patient room. In a few hours the patient had pain and the pupils were widely dilated. The vision was reduced to fingers and the T. was $+$ 1 in each eye. Patient was under eserine, each eye, since the first time she came to the hospital (March 21, 1918). The vision in the left eye was $\frac{4}{60}$ at this time and was not improved with lenses. The T. was full.

Discharged, May 24, 1918. R. V. equaled $\frac{6}{18}$; with a $-$ 2.00 sph. equaled $\frac{6}{12}$. Nasal field was contracted. O. D. was not cupped. T. was normal. Left eye. Vision was defective since 1917. T. full. O. D. was cupped and pale.

December 31, 1918, readmitted. January 3, 1919, right was trephined under cocaine. January 21st discharged. R. V. equaled $\frac{6}{36}$. Tn. Drainage was good. Nasal field almost obliterated. L. V. equaled no P. L. T. $+$ 1. Not painful.

Richard P., aged 44, married, transport worker, good health. Admitted, January 1, 1918, under Mr. Flemming. On admission right vision was $\frac{6}{36}$. The O. D. was not cupped. The T. was $+$. The left eye was quiet. Chamber was shallow. Pupil was active. Vision was $\frac{6}{6}$. Normal disk. Tn. Full field.

Discharged, January 4, 1918. Right eye was slightly injected. Pupil was pin-point. Anterior chamber was shallow. Tn. Given eserine for use at home. Full field. August, 1918, R. V. was $\frac{6}{6}$, L. V. was $\frac{6}{6}$.

Rosetta R., aged 72, widow, general health was fairly good. Admitted February 1, 1918, under Mr. Flemming. On admission she had acute congestive glaucoma in the left eye. Vision was hand movements. Fundus was obscured. T. + 1. February 1st had an iridectomy on left eye under $CHCl_3$.

February 12th discharged. Left cornea was bright. Iris pillars were free. A few vitreous opacities were present. Vision was $\frac{6}{60}$. Tn. Right eye was operated for chronic glaucoma at Moorfields Hospital, June 6, 1916 (iridectomy). Vision equaled $\frac{6}{24}$. O. D. was pale and cupped. T. was normal.

Georgine T., aged 35, married, pyorrhœa, general health good. Admitted December 6, 1918, under Mr. Flemming. On admission fundus was obscured. Right vision equaled fingers. T. was + 2. December 12th right trephined under cocaine.

December 20th, discharged. Right cornea was bright. Vision with a + 2.50 cyl. axis H. equaled $\frac{6}{18}$. Tn. Drainage was good. O. D. normal.

Left eye was quiet. Chamber was shallow. O. D. normal. Vision with a + 0.75 sph. and a + 2.00 cyl. equaled $\frac{6}{6}$. T. was normal.

Thomas P., aged 76, widower, good health. Admitted September 18, 1918, under Mr. Flemming. Patient attributed the onset of his eye trouble to worry. On admission the left eye showed a condition of acute congestive glaucoma. A large hyphæma was seen. September 20th an iridectomy was done under novocaine. The tension persisted and the eye was excised on October 10, 1918.

The right eye was normal. Vision equaled $\frac{6}{6}$. Tn. Full field. On pathological examination of the left eye I found the angle closed. The optic nerve was deeply cupped. No evidence of cyclitis was present.

Florence S., aged 56, married, subject to bilious attacks. Admitted, January 26, 1918, under Mr. Greeves. On admission the left eye was in a state of acute congestive glaucoma. Vision equaled fingers. T. was + 1. January 30, 1918, left trephined, under novocaine.

In the right eye the symptoms were less marked. The cornea was hazy and the pupil dilated. Vision equaled $\frac{6}{36}$. T. + 1. O. D. was not cupped. The right field showed about a 20 degree concentric contraction. February 13th, right trephined under cocaine.

February 27th, discharged. Left quiet. Vision was $\frac{3}{60}$.

O. D. was cupped and pale. Tn. Right eye was quiet. Vision equaled $\frac{6}{18}$. O. D. not cupped. Tn.

Alice P., aged 50, single, good health. Admitted, August 7, 1918, under Mr. Greeves with subacute glaucoma in each eye. Vision in each was $\frac{6}{60}$ and the T. +. August 10th left trephined and August 24th right trephined under novocaine.

Discharged, September 4th. Right cornea was bright. Pupil was dilated and circular (atropine in ward). Vitreous was cloudy. Vision equaled hand movements. Tn. O. D. not seen.

The left eye was quiet. Vision equaled $\frac{6}{36}$. O. D. normal. Tn.

December 4, 1918, R. V. equaled $\frac{6}{60}$. L. V. equaled $\frac{6}{6}$.

Nathan T., aged 53, clerk, married, good health. Admitted December 20, 1918, under Mr. Hudson, with subacute glaucoma in the left eye. On admission the pupil was pin-point (eserine). Vision was $\frac{6}{6}$. Shallow chamber. Full field. Tn.

Right quiet. Lens opacities. Vision was $\frac{6}{36}$. T. was full.

Discharged December 23d. T. normal in each eye.

January 8, 1919, began to see rainbows with right eye. Pupil was semi-dilated under eserine treatment. Vision $\frac{6}{60}$. January 27th right trephined. A bead of vitreous was lost at operation. Field showed 10 degrees' contraction above between fifteenth and ninetieth meridians.

February 6th, discharged. Right quiet. Vitreous was hazy. Vision was $\frac{6}{60}$. Tn. Left was quiet. Vision was $\frac{6}{6}$. Tn.

Mary B., aged 58, married, good health. Admitted October 9, 1918, under Mr. Fisher. Vision equaled fingers. T. was + 2. Fundus was obscured. No treatment prior to admission. October 15th left was trephined under cocaine.

Discharged, October 29th. Left was quiet. Vision was $\frac{6}{18}$. O. D. normal. Tn. Drainage was good. January 28, 1919, L. V. was $\frac{6}{6}$ with a + 0.50 Cyl. Right chamber was shallow. Normal disk. Field was full. Vision was $\frac{6}{6}$. Tn.

Clara B., aged 47, married, good health, munition worker. Admitted, November 13, 1918, under Mr. Fisher. Treated by her doctor with "drops." On admission vision equaled fingers. T. + 1. Fundus was obscured. November 15th a paracentesis was done—cocaine. November 29th right eye was trephined. A large piece of iris was excised—cocaine.

December 2d, discharged. R. V. was $\frac{6}{6}$. Full field. Tn.

L. V. was $\frac{6}{6}$. Chamber was shallow. Pupil, eccentric and sluggish. O. D. was normal. Tn. Full field.

July 8, 1919, R. V. with a + 1.50 sphere was $\frac{6}{6}$. O. D. normal.

Maria F., aged 67, married, good health. Admitted, November 6, 1918, under Mr. Flemming. On admission the fundus was obscured and the T. + 1. No treatment was given prior to November 5th, at which time she came as an out-patient.

November 8th an iridectomy was done under novocaine.

November 27th discharged. Cornea was bright. Pupil dilated and the nasal pillar was drawn up toward the scar above. Lens opacities were present and the vitreous was cloudy. O. D. not seen clearly. Vision equaled fingers. Tn. The eye was not injected.

Left eye was quiet. Chamber was shallow. Vision was $\frac{6}{18}$. Vision with a + 2.00 sph. was $\frac{6}{6}$. Full field. Tn.

April 29, 1919. Left eye was trephined under novocain, as the nasal field was becoming contracted and the vision was failing.

Discharged, May 6th. L. V. was $\frac{6}{12}$. O. D. not cupped. Tn. Good drainage.

Mary S., aged 41, single, dressmaker. Admitted, August 1, 1918, under Mr. Hepburn. Was operated for appendicitis; thrombosed veins in the right leg followed during the summer of 1918, otherwise has always been healthy. On admission the eye showed a condition of acute congestive glaucoma. August 6th a right paracentesis was done under cocaine. August 7th and 8th R. T. was +. August 8th an iridectomy was made under $CHCl_3$.

Discharged, August 18th. Right cornea was still hazy. Iris pillars were free. Anterior chamber was shallow. Vitreous was cloudy. Vision with a + 5.00 cyl. equaled $\frac{6}{24}$.

She had no treatment prior to admission.

L. V. with a + 3.50 sphere and a + 2.00 cyl. equaled $\frac{6}{6}$. T. was full. Treated with eserine.

Thomas W., aged 74, widower, good health. Admitted December 2, 1918, under Mr. Hepburn. March, 1918, contracted influenza and both eyes were inflamed three weeks. Lens opacities. R. V. equaled $\frac{6}{18}$. On admission the left pupil was dilated and inactive. Vision equaled fingers. T. + 1. December 5th an iridectomy was made on the left eye under $CHCl_3$. December 30th, discharged. Left was quiet. Cornea was hazy. Iris pillars were free. Lens opacities. Vision equaled fingers. Fundus obscured. Tn.

Right eye had lens opacities. Vision was $\frac{6}{18}$. Tn. The anterior chamber was shallow and the pupil reacted sluggishly.

Alice B., aged 72, widow, fair health. Admitted January 6, 1919, under Mr. Hepburn. Since January 2d, saw only light. On admission right eye was injected. Vision equaled P. L. T. was + 2. Fundus obscured. January 9th, had a right iridectomy under $CHCl_3$.

January 23d discharged. Right eye showed a slight degree of injection. Iris pillars were free. Vitreous was cloudy. Vision equaled fingers. Tn. Retina detached.

Left eye. Vision was $\frac{6}{18}$; with a + 3.00 sph. equaled $\frac{6}{6}$. Chamber was shallow. Tn. Full field.

Julia W., aged 49, married, good health. Admitted, January 7, 1919, under Mr. Hepburn. Treated by her doctor with "drops." On admission vitreous was cloudy. Vision with a + 1.50 sph. equaled $\frac{6}{18}$. Tn.

January 9th, left iridectomy under $CHCl_3$.

January 23d, discharged. Left eye was quiet. Cornea was bright. Vision with a + 2.50 sph. and a + 1.00 cyl. H. was $\frac{6}{18}$. O. D. not cupped. Tn.

Right chamber shallow. Pupil circular and active. Vision $\frac{6}{6}$. Full field. Tn.

February 10, 1919, L. V. with correction equaled $\frac{6}{6}$.

Emily W., aged 49, married, laundress, good health. Admitted February 4, 1918, under Mr. Hepburn. On admission both eyes had subacute glaucoma. L. V. equaled fingers. T. + 1. Fundus obscured. R. V. = $\frac{6}{60}$

February 15th, left had a paracentesis under cocaine.

February 18th, left was trephined, under cocaine. February 20th, left anterior chamber was full of blood, and was very painful. T. was normal.

March 7th, right was trephined.

March 28th, discharged. Left was quiet. Cornea was clear. Hyphæma. Vision was $\frac{6}{24}$. O. D. was cupped. Tn. Right was also quiet. Vision equaled $\frac{6}{60}$. O. D. was cupped and pale. Tn. March 13, 1919, L. V. was $\frac{6}{6}$ unaided. Tn. O. D. slightly cupped.

Julia A., aged 53, widow, good health. Admitted, December 31, 1918, under Mr. Hepburn. Treated with eserine drops by her family physician. On admission the left cornea was hazy. The chamber was shallow and the pupil contracted. Vision was $\frac{4}{60}$. Left field showed about a 25 degree nasal contraction. T. + 1. O. D. cupped and pale.

January 2, 1919, left trephined under novocaine.

January 17th, discharged. Cornea was bright. Vision was $\frac{6}{60}$. O. D. was cupped. Tn.

Right was quiet. Shallow chamber, normal fundus. Vision equaled $\frac{6}{9}$. Tn. Full field.

April 10, 1919, L. V. with a -5.00 sphere and a -0.75 cyl. equaled $\frac{6}{18}$.

Thomas P., aged 74, widower, salesman, good health. Admitted March 3, 1919, under Mr. Hepburn. Present attack in right eye began February 26th. No treatment. On admission there was ptosis of each lid. The right eye showed a condition of acute congestive glaucoma. Vision was hand movements. Fundus was obscured. T. + 2.

March 4, 1919, right iridectomy was made under $CHCl_3$.

March 17th, discharged. Cornea was bright. Iris pillars were free. Lens opacities obscured the O. D. Vision equaled fingers. Tn.

Left eye. The chamber was shallow. Vision was $\frac{6}{12}$. T. full. Field was not contracted. O. D. normal.

Robert F., aged 57, married, gas-fitter, good health. Admitted March 5, 1919, under Mr. Moore. On admission the left eye showed a state of acute congestive glaucoma. No treatment prior to admission. The vision equaled fingers. T. + 1. Fundus was obscured.

March 5, 1919, left iridectomy—$CHCl_3$.

March 15th, discharged. Cornea was bright and the iris pillars were free. Vision with a $-.75$ sph. and a + 3.00 cyl. axis H. was $\frac{6}{24}$. O. D. normal. Tn.

Right eye was operated in 1914 for chronic glaucoma at Moorfields Hospital. Vision was $\frac{6}{18}$. O. D. was cupped. Iris pillars were free and the Tn.

Elizabeth E., aged 60, married, good health. Admitted, February 5th, 1918, under Mr. Moore with acute congestive glaucoma in the left eye, of twenty-four hours' duration. Had a similar attack five weeks before, but less severe. No treatment. Vision equaled fingers. Fundus was obscured. T. + 2.

February 26th, a left iridectomy was done under $CHCl_3$.

March 12th, discharged. Left cornea was bright. Wide iridectomy. O. D. normal. Vision was $\frac{6}{6}$. Tn.

Right vision was $\frac{6}{12}$; with a + 3.50 sphere was $\frac{6}{6}$. Nasal field was contracted about 10 degrees between fifteenth and seventy-fifth meridians.

George T., aged 74, single, fairly good health. Admitted May 20, 1919, under Mr. Flemming, with acute congestive

glaucoma in the right eye. Duration was six days. Treated with "drops and lotion." Vision was reduced to P. L. T. was + 2.

May 30, 1919, an iridectomy was made under novocaine.

June 24th, discharged. Right eye was still injected. No keratitis punctata was seen. Iris pillars were free. Much lens opacity. Fundus obscured. Vision equaled P. L. with projection quick and accurate. Tn.

Left eye. The chamber was shallow. The pupil acted well. Lens opacities obscured the fundus details. Vision equaled $\frac{6}{18}$. T. full.

Edgar F., aged 42, married, good health, baker. Admitted June 17, 1918, under Mr. Hudson.

The right vision failed suddenly and when he came to the out-patient department the right eye was in a state of acute congestive glaucoma. The vision was reduced to P. L. and the T. was + 2. There was a defect in the lower temporal field, although this was difficult to outline owing to his poor sight.

June 17th the right eye was trephined under novocaine. The iris bled freely when the small iridectomy through the trephine opening was made.

June 27th was discharged. The right eye was still injected but there were no deposits on the cornea and no synechiæ were seen. The fundus was dimly seen. Vision equaled no P. L. Tn. Good drainage.

Left eye. Vision $\frac{6}{9}$. The chamber was shallow. Tn.

Edith B., aged 63, single, good health. Admitted June 23, 1919, under Mr. Worth. First attack of obscured vision and halos was in May, 1919, in the left eye. No treatment. Present attack began a few days before admission. Fundus details were obscured. Vision equaled $\frac{3}{60}$. T. + 1.

June 26, 1919, left was trephined under novocaine.

Discharged July 10th. Left eye was quiet. Vision equaled $\frac{6}{24}$. O. D. was cupped but the color was good. Tn. Good drainage.

Right eye. Vision $\frac{6}{60}$; vision with a + 6.00 sph. was $\frac{6}{12}$. Chamber was shallow. Pupil active and circular. Tn. O. D. was not cupped.

Annie G., aged 53, married, pyorrhœa, varicosed veins, otherwise healthy. Admitted, June 16, 1917, under Mr. Parsons. No treatment. On admission left eye was in a state of acute congestive glaucoma. Vision equaled fingers. Fundus was obscured. T. + 1.

June 20th left iridectomy was made under $CHCl_3$.

July 5th discharged. Left cornea was bright. Iris pillars were free. Vision was $\frac{6}{24}$. Vitreous hazy. O. D. not cupped. Tn. The left field showed about 20 degrees nasal contraction.

Right eye. Chamber shallow. Pupil active and circular. Normal fundus. Vision $\frac{6}{18}$. Field full.

Rose C., aged 50, married, good health. Admitted, September 1, 1917, under Mr. Parsons. No treatment. On admission the right cornea was hazy. Anterior chamber was shallow. Pupil was dilated and inactive. Vision equaled $\frac{6}{18}$; not improved with lenses. O. D. was not cupped. T. + 1.

September 8, 1917, an iridectomy was made under $CHCl_3$.

September 29th, discharged. Right cornea was clear. Pillars were free. Chamber was shallow. Small hyphæma. Vision was $\frac{6}{60}$. O. D. was slightly cupped.

Henry G., aged 65, married, fairly good health. Admitted, September 5, 1917, under Mr. Parsons. No treatment. On admission the right cornea was hazy. Much engorgement of the ciliary vessels was present, and the pupil was dilated and fixed. Lens opacities. Vision was reduced to fingers. Six hours after eserine was used vision equaled $\frac{6}{36}$. September 8th O. D. was seen and no cupping was present.

September 15th, right trephined under cocaine.

The left eye showed a smilar state on admission. The vision was $\frac{6}{60}$ and six hours after treatment was $\frac{6}{12}$. The optic disk was not cupped.

September 18th, left eye was trephined—cocaine.

Discharged, September 22d. R. V. equaled $\frac{6}{36}$. O. D. not cupped. Right field showed about 25 degrees contraction of the nasal field. L. V. equaled $\frac{6}{9}$. Nasal field contracted above about 10 degrees. Lens opacities were seen at the periphery of the lens.

Elizabeth P., aged 63, married, good health. Admitted, under Mr. Parsons, January 24, 1917. Came as an outpatient January 20th and refused to be operated. Given eserine ½%. Right eye inflamed and painful in December, 1916. On admission right eye showed a condition of acute congestive glaucoma. Vision equaled hand movements. T. + 1. Left eye was painful two days, on admission, and showed a similar condition as right eye. Vision equaled fingers. T. + 1. Although she used eserine and hot applications at home the left eye was little benefited.

January 24, 1917, an iridectomy was done under $CHCl_3$.

Discharged, February 7th; left was injected. No keratitis punctata seen. Hyphæma. Pillars were free. Vision $\frac{6}{36}$. Tn. Refused operation on right. The right cornea was hazy. Vision equaled hand movements. T. +. Pupil semi-dilated. Vitreous hazy. O. D. obscured.

August 9, 1919. L. V. equaled $\frac{6}{18}$. O. D. was not cupped. R. V. was $\frac{6}{36}$. O. D. was cupped.

Eliza D., aged 68, married, good health. Admitted February 2, 1917, under Mr. Parsons. Left eye first inflamed in January, 1917. No treatment. On admission the left eye showed a condition of acute congestive glaucoma. Vision equaled fingers. Fundus was obscured. T. + 1.

The right eye first became inflamed four days after the left. There was no history of emesis or halos. On admission the eye was much injected and very painful. It showed a similar condition to the left. Vision equaled hand movements. T. + 1. Fundus was obscured.

February 3d, right and left iridectomy under $CHCl_3$.

February 21st patient injured his right eye which was followed by marked ciliary injection.

Discharged, March 3, 1917. Left cornea was still hazy. Nasal pillar drawn up to incision above. No keratitis punctata. Fundus details not seen clearly. Tn. Vision equaled fingers. Lens opacities obscured the fundus.

Right eye. Cornea was distinctly hazy. No deposits on posterior surface. Iris pillars were free. No synechiæ were seen. Much lens opacity. Vision equaled P. L. No inflammatory deposits were present in the coloboma area or on the lens capsule. The fundus was obscured. Tn.

Lydia McC., aged 60, widow, good health. Admitted November 2, 1917, under Mr. Flemming. Left was first inflamed October 20th. On admission the left eye showed a condition of acute congestive glaucoma. Vision equaled hand movements. T. + 2. Fundus was obscured.

November 2, 1917, left iridectomy was made under $CHCl_3$.

November 27th discharged. Left was quiet. Iris pillars were free. Small hyphæma was present. Vitreous hazy. Vision equaled $\frac{4}{60}$. O. D. slightly cupped. Tn.

Right quiet. Chamber was shallow. Vision was $\frac{6}{24}$; vision with a + 3.00 sphere was $\frac{6}{6}$. Tn. January, 1919, R. V. equaled $\frac{6}{6}$ with above glass and J. 1 with a + 5.00 sphere. L. V. equaled $\frac{6}{24}$.

Elizabeth R., aged 66, married, pyorrhœa, good health. Admitted, May 17, 1917, under Mr. Flemming. No treatment. On admission the left eye showed a condition of sub-

acute glaucoma. Pupil was small (eserine from out-patient department) and round. Chamber was shallow. Lens opaque. Vision equaled fingers. T. + 1.

May 18th left iridectomy was made under novocaine.

May 21st patient struck her left eye with her hand, which was followed by a hyphæma.

June 5th, discharged. Left eye was quiet. Cornea was bright. No keratitis punctata. Iris pillars were free. Lens opacity obscured the fundus. Vision equaled fingers. Projection was accurate and quick. Tn. Hyphæma was absorbed.

Right eye was quiet. Vision was $\frac{6}{6}$. Chamber was shallow. Pupil was circular and active. Tn. O. D. normal.

Emma L., aged 68, widow, pyorrhœa, fair health. Admitted July 17, 1917, under Mr. Flemming. No treatment. On admission the left eye was injected. There was a central corneal haze. The chamber was shallow. The pupil dilated and fixed. Lens opacities obscured the fundus. Vision equaled P. L. T. + 1.

July 17th left iridectomy was made under $CHCl_3$. The eye became infected and was eviscerated July 23d.

Discharged, August 13th. Right eye was quiet. Chamber was shallow. Pupil was active and circular. Vision was $\frac{6}{36}$; vision with a − 1.50 sph. equaled $\frac{6}{6}$. O. D. normal. Tn.

Phœbe E., aged 49, married, general health good. Admitted April 6, 1916, under Mr. Hudson. No treatment. On admission the right cornea was hazy and very œdematous. The chamber was shallow, and the pupil dilated and inactive. Vision equaled $\frac{6}{60}$. T. + 1.

April 6th right iridectomy was made under $CHCl_3$.

April 17th, discharged. Cornea was bright. Iris pillars were free. Vitreous was hazy. O. D. not cupped. A few small hemorrhages were seen near the disk. Vision with a − 3.00 sph. and a + 6.00 cyl. axis H. equaled $\frac{6}{12}$. Left eye was quiet. Vision was $\frac{6}{6}$ unaided. Shallow chamber. T. with Schiotz was 25*mm* Hg.

Annie F., aged 63, widow, good health. Admitted, August 4, 1917, under Mr. Spicer. Right eye was first inflamed in July, 1917, while in a hospital for an injury to head. No eye treatment. On admission to Moorfields Hospital the right eye showed a condition of acute congestive glaucoma. Vision equaled fingers. Fundus was obscured. T. + 1.

August 5th a right paracentesis was done under $CHCl_3$.

August 7th an iridectomy was made under $CHCl_3$.

August 24th, discharged. Right cornea was bright. Iris

pillars were free. Vitreous slightly hazy. Vision equaled $\frac{6}{24}$. Tn.

Left quiet. Vision $\frac{6}{6}$. Chamber shallow. Tn.

Elizabeth G., aged 52, married, good health. Admitted August 14, 1917, under Mr. Spicer. Right eye was first inflamed August 7, 1917. No treatment. On admission the eye showed a condition of acute congestive glaucoma. Vision equaled fingers. T. + 2.

August 15th an iridectomy was made under $CHCl_3$.

August 24th, discharged. Right cornea was bright. Iris pillars were free. Vitreous was hazy. Vision equaled $\frac{4}{60}$. Tn. O. D. was not cupped.

Left eye showed a condition of absolute glaucoma. Vision equaled no P. L. T. + 2.

May, 1919, R. V. equaled $\frac{6}{12}$.

Harriet S., aged 50, single, good health. Admitted, September 10, 1916, under Mr. Lawford. No treatment.

Present attack began two days before she was admitted On admission the right cornea was hazy, the chamber shallow, and the pupil was dilated. Vision equaled fingers. T. was + 1.

September 12th an iridectomy was made under $CHCl_3$ and ether.

October 4th, discharged. Right cornea was hazy. Iris pillars were free. O. D. was not cupped. Vision equaled $\frac{6}{36}$. Tn.

Left eye. Anterior chamber was shallow. Pupil small (eserine); vision was $\frac{6}{12}$. O. D. was normal. Tn.

Eliza B., aged 69, widow, good health. Admitted September 8, 1917, under Mr. Lawford. In April, 1917, first saw halos, but the right eye never was painful until September 3d, five days before admission. No treatment. On admission the right eye showed a state of acute congestive glaucoma. The fundus was obscured and the vision equaled fingers. T. was + 2.

September 12th an iridectomy was done under novocaine.

October 3d, discharged. Right cornea was almost clear. Iris pillars were free. Vitreous was hazy. O. D. was not cupped. Tn. Vision equaled $\frac{6}{36}$. Nasal field was not contracted.

Left eye. Chamber was shallow. Vision equaled $\frac{6}{6}$. Full field. Tn.

September 5, 1918. R. V. equaled $\frac{6}{6}$.

Annie C., aged 59, married, myxœdema. Admitted,

September 28, 1917, under Mr. Lawford. No treatment. Present attack began twenty-four hours before she was admitted. On admission the left eye was not very painful. The cornea was steamy and insensitive. The chamber was shallow. The pupil was dilated and inactive. Vision equaled fingers. T. + 1.

September 29th an iridectomy was made under novocaine.

October 20th, discharged. Left quiet. Iris pillars were free. Vitreous was hazy. Vision equaled $\frac{4}{6}$. Tn. O. D. was cupped.

Right eye. Chamber was shallow. Vision equaled $\frac{6}{6}$. O. D. was normal. Tn.

Jane B., aged 72, married, subject to rheumatism. Admitted, August 17, 1917, under Mr. Lawford. July 29, 1917, patient had her first attack of pain in right eye, accompanied by several attacks of emesis. Treated by her family physician with "drops." Was given eserine August 15th at a London hospital. On admission to Moorfields Hospital the right eye showed a state of acute congestive glaucoma. Vision equaled $\frac{3}{6}$. T. + 1.

August 18th an iridectomy was made under $CHCl_3$.

September 5th discharged. Iris pillars were free. Vitreous was hazy. Tn.

Left eye. Chamber was shallow. Vision was $\frac{6}{6}$. O. D. was normal. Tn. Field was full.

September 21, 1918, R. V. equaled $\frac{6}{6}$. O. D. was normal. The nasal field was contracted between the vertical and sixtieth meridians about 10 degrees.

Elizabeth O., aged 71, single, good health. Admitted, July 11, 1917, under Mr. Lawford. First saw halos in May, 1917. Present attack began July 9th. No treatment. On admission the right eye showed a state of acute congestive glaucoma. Vision equaled fingers. T. was + 2.

July 14th an iridectomy was made under $CHCl_3$.

August 1st, discharged. The iris pillars were free. Vitreous haze obscured the fundus details. Vision equaled $\frac{6}{6}$. Tn.

Left eye. Operated for subacute glaucoma at Moorfields Hospital, February 10, 1915. Vision equaled $\frac{4}{6}$. O. D. was cupped.

Joseph W., aged 68, married, good health. Admitted October 22, 1917, under Mr. Hepburn.

October 14th the right eye was red, but not very painful. No treatment. On admission the right cornea was hazy, the chamber shallow, and the pupil dilated. Vision was $\frac{6}{6}$. The fundus was apparently normal. T. + 1.

October 23d an iridectomy was made under $CHCl_3$.

October 25th patient was frightened during an air raid and got up from his bed. In the morning the nasal pillar was prolapsed.

Iris pillar was replaced with a repositor and remained in place.

November 1st discharged. Right eye was quiet. Iris pillars were free. Vision was $\frac{6}{15}$. Tn. O. D. not cupped.

Left eye. Chamber was shallow. Vision was $\frac{6}{9}$. O. D. was normal. Tn. Nasal field was contracted about 30 degrees, except below.

Sarah K., aged 61, widow, good health. Admitted, August 7, 1917, under Mr. Fisher, with subacute glaucoma in left eye. On admission the left eye showed the cornea was hazy, the chamber shallow, and the pupil dilated. Vision was reduced to fingers. T. was + 2. Fundus was obscured.

August 10th an iridectomy was made under $CHCl_3$ down and out.

August 28th, discharged. Iris pillars were free. Vitreous was hazy. Vision was $\frac{6}{60}$. Tn. O. D. normal. Macular changes.

October 8th, readmitted on account of tension. The cornea was slightly hazy. Vision was $\frac{6}{60}$. O. D. was not visible.

October 9th left trephined under cocaine.

October 23d, discharged. Cornea was bright. Vision was $\frac{6}{60}$. O. D. was cupped. Good drainage. Tn.

Right eye showed old choroidal changes at the macula. Vision was $\frac{4}{60}$. Tn. O. D. was not cupped. The chamber was shallow.

Sarah N., aged 48, married, pyorrhœa, good health. Admitted, November 9, 1917, under Mr. Fisher. Right vision misty since May, 1917. No treatment. On admission the right eye showed a condition of subacute glaucoma. Vision equaled fingers. Fundus was obscured. T. + 1.

November 11th right was trephined under cocaine.

November 27th discharged. Anterior chamber was very shallow. Vision was $\frac{6}{36}$. Vitreous hazy. O. D. was cupped. Tn.

Left eye. The anterior chamber was shallow. Vision was $\frac{6}{9}$. O. D. was normal and the Tn. Full field.

William C., aged 57, married, good health. Admitted, November 5, 1917, under Mr. Collins with an attack of acute congestive glaucoma in each eye. Present attack began five days before admission. The attack in the left eye began

twenty-four hours prior to admission and was accompanied by emesis. No treatment.

On admission the eyes showed acute congestive glaucoma. The vision in each was reduced to fingers. T. was + 1.

November 5th right and left were trephined under $CHCl_3$.

November 22d, discharged. Right eye was injected. Fundus obscured by the hazy vitreous. Vision equaled $\frac{2}{60}$. Tn. Drainage was not satisfactory. (Disk fell into anterior chamber.)

Left eye was quiet. The cornea was bright. Vision was $\frac{6}{24}$. O. D. not cupped. Full field. Tn.

Lavater W., aged 62, married, good health. Admitted December 10, 1917, under Mr. Collins with an attack of acute congestive glaucoma in the right eye of five days' duration. No treatment. Vision equaled shadows. T. was + 1.

December 10th right trephined—novocaine.

December 31st, discharged. Right was injected, and the cornea hazy. Vision equaled fingers. Lens opacity obscured the fundus. Tn.

Left eye was blind since October, 1917, the result of an injury.

Lydia H., aged 78, married, general health was not good. Admitted, October 18, 1916, under Mr. Collins. The right eye was occasionally painful for some time. During the summer of 1916 the sight was dim at times. No treatment prior to admission. The cornea was hazy, the chamber shallow, the pupil dilated and inactive. O. D. and fundus details were obscured. T. + 1.

October 19, 1916, right eye was trephined under cocaine.

October 28th a large detachment of choroid was seen at the temporal side. Also a small hemorrhage near the disk.

November 8th detachment was extending upward. The anterior chamber was very shallow. Vision with a + 4.00 sphere was $\frac{6}{18}$.

November 25th. Tn. No iritis. Atropine ordered and the tension was +, twenty-four hours later. No detachment of choroid seen. Atropine discontinued.

December 20th T. +. No keratitis punctata and no synechiæ were seen. Vision equaled $\frac{1}{60}$.

December 21, 1916, a second trephine was necessary.

Discharged, January 8th. Cornea bright. No deposits on posterior surface. No detachment seen. Vision equaled hand movements. Hyphæma, and blood in pupil area obscured the fundus.

Left eye was blind from glaucoma. Vision equaled no P. L.

Eliza S., aged 61, married, good health. Admitted January 11, 1917, under Mr. Hudson. December 21, 1916, the right vision failed over night. The left vision failed a few days later. No treatment.

On admission both eyes were in a state of acute congestive glaucoma. The vision equaled fingers. T. was + 2.

January 11th an iridectomy was made on each eye under $CHCl_3$.

January 25th discharged. The corneæ were bright in each eye. The iris pillars were free. No keratitis punctata was seen and both lenses were uninjured. In each eye the vitreous was hazy. Both disks were slightly cupped. Vision in each was $\frac{6}{18}$. Tn. June 30, 1917, R. and L. V. equaled $\frac{6}{9}$. Tn.

Sarah M., aged 72, widow, health fairly good. Admitted April 23, 1917, under Mr. Collins. April 17th the right eye became inflamed and painful. No treatment prior to admission.

April 23d the right eye was slightly injected. The pupil was oval and inactive. Central lens opacity. Vision was $\frac{6}{18}$. Fundus details obscured. T. + 1. Nasal field was contracted one half. Saw halos the first time in 1916.

April 24th right trephined under novocaine.

Discharged May 10th. R. V. was $\frac{6}{18}$. Central lens opacity. O. D. was cupped. Drainage was good. Tn.

Left eye had chronic glaucoma. Vision equaled hand movements. Lens opacity obscured the fundus. T. + 1. Was not painful. Eye was quiet.

Bessie W., aged 45, single, good health. Admitted December 25, 1915, under Mr. Lawford. Was under treatment by her doctor who gave "drops" for the pain. On admission the left eye showed a condition of acute congestive glaucoma. Vision equaled hand movements. T. + 2.

December 25th an iridectomy was made under $CHCl_3$ and ether.

January 8, 1916, discharged. Left cornea was bright. Iris pillars were free. O. D. was not cupped. Vision was $\frac{6}{18}$. Tn.

Right eye. Vision was $\frac{6}{9}$. Shallow chamber. Tn.

George P., aged 79, married, laborer, fair health. Admitted July 25, 1916, under Mr. Parsons. July 4, 1916, the right eye first became inflamed. No treatment. On admission the eye showed a condition of subacute glaucoma. Vision equaled $\frac{6}{18}$. T. +.

July 26th right trephined under novocaine.

August 9th discharged. Right showed lens opacity (nucleus). O. D. not cupped. Vision equaled fingers. Tn. Good drainage.

Left eye. Vision was $\frac{6}{6}$. Lens opacities. Full field. Tn. O. D. was not cupped.

George R., aged 48, blacksmith, good health. Admitted September 20, 1916, under Mr. Parsons. Left eye was first inflamed and painful twenty-four hours before admission. On admission the left eye showed a state of acute congestive glaucoma. Vision equaled $\frac{6}{60}$. T. + 1. Fundus details were obscured.

September 27th left Tn. Patient refused an operation. Discharged.

November 29th readmitted on account of a second attack in left eye. Patient had omitted to use his eserine drops as the eye felt all right. On admission the left eye showed a severe glaucomatous attack. The vision equaled hand movements. The pupil was widely dilated and the pain was very intense.

December 6th left iridectomy under cocaine.

December 15th discharged. Left eye was quiet. Iris pillars were free. Lens clear. Vision was $\frac{6}{6}$. Tn.

Right eye. Operated for acute glaucoma in 1914 at Moorfields Hospital. Vision equaled $\frac{6}{6}$. O. D. normal. Tn.

Edward F., aged 71, salesman, fair health. Admitted March 16, 1916, under Mr. Hudson. Left painful since March 9th. On admission the left eye showed a state of subacute glaucoma. Vision equaled fingers. T. was + 2. Fundus obscured.

The right eye showed a state of chronic glaucoma. Vision equaled P. L. T. + 2.

March 16th right and left were trephined under cocaine.

Discharged April 11th. Left cornea showed much striated opacity. O. D. not cupped very much, color was good. Tn. Vitreous hazy. Vision equaled $\frac{6}{60}$.

Mary R., aged 68, widow, fair health. Admitted April 10, 1916, under Mr. Hudson. Was treated for "iritis" with atropine drops. In February patient was ill with influenza. Present attack began a few days before admission. On admission both eyes showed a state of subacute glaucoma. The vision in each was less than $\frac{4}{60}$.

April 11th a right and left iridectomy was made under $CHCl_3$ and ether.

May 2d discharged. Left choroid detached. Vision was

shadows. Tn. Right inner pillar caught up. O. D. cupped. Vision was reduced to fingers. Tn.

Emma T., aged 60, married, good health. Admitted May 1, 1916, under Mr. Hudson. Right vision failed in April, 1916, during an attack of influenza. On admission the right eye showed a condition of subacute glaucoma. Vision equaled fingers. T. + 1.

May 1st right paracentesis under cocaine.

May 4th right trephined under cocaine and novocaine.

May 15th discharged. Right quiet. Vision was $\frac{6}{18}$. Tn. Nasal field was contracted and blind spot enlarged.

Left quiet. Chamber was shallow. Vision equaled $\frac{6}{12}$. Tn. O. D. was not cupped. Full field.

Stephen P., aged 60, married, fair health. Admitted January 1, 1916, under Mr. Spicer. Right vision was misty in 1912 and since at intervals. On admission the right eye was in a state of acute congestive glaucoma. Vision was reduced to fingers and the T. was + 1.

January 7th an iridectomy was made under $CHCl_3$.

January 20th discharged. R. V. equaled $\frac{6}{9}$. Pillars were free. Tn.

Left cornea was bright. Pupil contracted (eserine). Tn. Vision was $\frac{6}{9}$.

Arthur G., aged 51, married, good health. Admitted June 29, 1916, under Mr. Hudson. Left eye was first inflamed twenty-four hours before admission. No treatment. On admission the left eye showed a state of acute congestive glaucoma. Vision equaled fingers. T. was + 1.

June 30th an iridectomy was done under $CHCl_3$.

July 8th discharged. Cornea was bright. Iris pillars were free. Vision equaled $\frac{6}{18}$. O. D. was not cupped. Tn.

Right eye was operated for glaucoma at Moorfields Hospital in 1914. Vision was $\frac{6}{12}$. O. D. was not cupped. Tn.

Sarah C., aged 49, married, good health. Admitted December 7, 1916, under Mr. Spicer. Right eye was first inflamed and painful November 29, 1916. No treatment. On admission the right eye showed a state of acute congestive glaucoma. Vision equaled fingers. T. was + 2.

December 8, 1916, an iridectomy was made under $CHCl_3$.

December 24th discharged. Right was quiet. Iris pillars were free. Anterior lens opacities. O. D. not cupped. Vision was $\frac{6}{60}$. Tn.

Left eye. Had chronic glaucoma. Vision equaled no P. L. T. + 2. May 4, 1917. R. V. equaled $\frac{6}{18}$.

Emma R., aged 63, single, good health. Admitted January 4, 1916, under Mr. Flemming. Taken direct to theater and had an iridectomy on left eye under $CHCl_3$.

On admission the eye showed a state of acute congestive glaucoma. Vision equaled fingers. Fundus details obscured but O. D. was not cupped. T. was + 1.

January 19th discharged. Cornea was bright. Iris pillars were free. Vitreous opacities. Posterior polar cataract. O. D. not cupped. Vision with a + 2.00 cyl. was $\frac{6}{18}$.

Right quiet. Vision was $\frac{6}{18}$. Posterior polar cataract. T. +. O. D. was not cupped.

Evelyn E., aged 45, married, good health. Admitted November 14, 1916, under Mr. Flemming. No treatment. The left eye showed a state of acute congestive glaucoma on admission. Vision equaled fingers. T. + 2.

November 15th an iridectomy was made—$CHCl_3$.

November 23d vision was $\frac{6}{36}$. Much striate corneal opacity.

November 30th the T. was +. Cornea was œdematous.

December 1st left paracentesis was necessary, under novocaine.

December 2d left anterior sclerotomy under $CHCl_3$. Several recent hemorrhages were seen about the disk, which was slightly cupped. Vision with a + 4.00 cyl. Vision equaled $\frac{6}{12}$. Tn.

December 22d discharged. Cornea was slightly œdematous above. No keratitis punctata or synechiæ present. Iris pillars were both drawn up slightly. T. +. Vision equaled $\frac{6}{12}$ with the same correction. O. D. slightly cupped. Many small hemorrhages about the disk.

Right eye trephined May 4, 1917.

July, 1919. R. V. was $\frac{6}{6}$. O. D. was normal. L. V. was $\frac{6}{12}$ with a + 4.50 cyl. Tn. O. D. was a good color.

John K., aged 70, married, fair health. Admitted February 4, 1916, under Mr. Hepburn. Left was first inflamed in January, 1916. No treatment. On admission the left eye showed a state of acute congestive glaucoma. Vision equaled fingers. T. was + 2.

February 5th an iridectomy was made under $CHCl_3$.

February 23d discharged. Left eye was slightly injected. Iris-pillars were free. Much lens opacity. Fundus was obscured. Vision equaled $\frac{3}{60}$. Tn.

Right eye was operated for glaucoma at Moorfields.

March 5, 1912 (iridectomy). Vision equaled $\frac{6}{24}$. O. D. was cupped.

William C., aged 65, married, fair health. Admitted May 10, 1916, under Mr. Hepburn with acute congestive glaucoma in right eye of two days duration. No treatment. Vision equaled fingers. T. was + 2.

May 11th an iridectomy was made under $CHCl_3$.

April 18th choroid was detached. Vitreous opacities.

May 1st no detachment of choroid was seen. Macular changes were seen, however.

May 5th discharged. Right was quiet. No detachment present. Iris pillars were free. Vitreous much clearer. Vision was $\frac{6}{60}$. O. D. was slightly cupped. No hemorrhages seen. Tn.

Left eye. Quiet. Vitreous opacities. No keratitis punctata. *Macular changes.* Vision equaled $\frac{6}{60}$. Vision with a + 3.00 sph. equaled $\frac{6}{12}$. O. D. was not cupped Tn.

March 10, 1918 left glaucoma iridectomy $CHCl_3$. L. V. on April 18, 1918, equaled $\frac{6}{6}$ with a + 2.00 sph. and a + 2.00 cyl. axis H. The left eye developed acute congestive glaucoma two days before admission accompanied by severe pain and vomiting.

Remarks: In the 87 patients admitted there were 103 eyes with acute or subacute glaucoma. The cases with chronic glaucoma in the other eye were merely mentioned with a note of the vision and condition of the optic nerve. The right eye was involved in 48 and the left in 55 cases. There were 24 males and 63 females.

Regarding the age; one case was 25, two were 31, twenty were between 40 and 50, twenty-one were between 50 and 60, thirty-seven between 60 and 70, fourteen between 70 and 80 and one between 80 and 90 years. A few of the cases were bilateral.

Influenza was present or preceded the attack in four cases, rheumatism in four, and three cases had pyorrhœa very badly. In two cases there was a history before the attack set in, and five cases showed a postoperative injury. A hyphæma developed in 8 cases. In only one case was the iris pillar prolapsed and it remained in place after the use of an iris repositor.

The cases admitted without treatment numbered 61. Boric lotion was used by 13, atropine by 4, and eserine by 8 of the cases. Previous attacks were noted in 74 of the cases, while 26 came to the hospital during or just after the first attack, 65 patients saw halos and 46 vomited with their attacks. A few were nauseated, but did not vomit.

In Stock's cases atropine was used on the second day. A mild

iritis was seen more frequently than after iridectomy. Stock advocated the removal of a hyphæma at the time of operation to aid healing. In all his cases he reduced the tension to 26 or less except in one. He did not encounter a single case of expulsive hemorrhage, loss of vitreous, or glaucoma fulminans. In 4 cases he was able to compare the tension after iridectomy with that of trephining, in the same or the fellow eye. In case 1, the tension after iridectomy was 33, after trephining 18. In case 2, after iridectomy 26, after trephining 22. In case 3 the tension after iridectomy was 95, after trephining 18 and in case 4, the tension was 26 as compared with 18 after trephining.

Stock included as acute all cases with a history of clouded vision or pain or halos.

Acute congestive glaucoma was present in 69 of the cases and subacute in the balance. Of the 63 iridectomies, the iris pillars were drawn up to the wound in 4, not including the one that prolapsed, mentioned above. In the 35 trephined cases, the disk fell into the anterior chamber in 1, without causing symptoms of iridocyclitis. A Herbert's sclerotomy was performed in 1 case and 1 case refused operation.

The fundus was obscured by lens opacities in 7 cases, while vitreous opacities obscured the details in 11 cases. In many of the latter, however, the fundus details were seen at a later date and no pathological cupping was noted. In 1 case the eye converged 20 degrees.

In 2 cases the choroid was detached, but only for a few days. Retinal detachment occurred in 2 cases and persisted. Retinal hemorrhages were present in at least 3 of the cases, and in each, near the optic disk. In 16 cases the optic disk showed cupping. No cupping was seen in 75, while in the remaining cases the fundus was obscured. Vitreous opacities were seen in 33 of the cases, although in many, the opacities were fine and disappeared later.

The nasal field in 11 cases was contracted from 5 to 30 degrees. Two cases showed a concentric contraction, one of 10 and the other of 20 degrees. In 4 cases no contraction was present, while in the remaining cases no field was obtained owing to the reduced vision at the time of entrance to hospital. I have not been able to secure the figures, regarding the fields of those cases, that had their fields charted since operation.

The lowest tension prior to operation, recorded with the Schiotz tenometer, registered *25mm Hg* and the highest 42, although in many of the operated cases the tension was undoubtedly much higher than this figure. In 9 cases the fellow eye had been operated for chronic glaucoma and in 3 for acute at some previous period.

In a review of the literature of the past ten years, covering the period since the modern decompression operations were introduced by LaGrange and Elliot, I have considered only those authors, who have reported ten or more cases. While many oculists have had this number or more, they have not published the details of their cases, so that a comparison could be made, and one has to confine his comparisons, with the cases reported by Stock and Meller. In a recent letter from Colonel Elliot asking him for his statistics to compare with other authors and those of the Moorfields Hospital, he was unable to furnish the same.

The following table gives a comparison of Stock, Meller, and the author, showing the visions on discharge, with the cases that were improved, remained stationary, or became worse. Those cases included under the title of author were operated by the following ophthalmic surgeons—Treacher Collins, Holmes Spicer, J. B. Lawford, Percy Flemming, J. Herbert Parsons, J. Herbert Fisher, Claude Worth, Malcolm Hepburn, A. C. Hudson, and Foster Moore.

AUTHOR.				S. M. A.	S. M. A.	S. M. A.
CASES.			VISION.	IMPROVED.	STATIONARY.	WORSE.
Stock.	*Meller.*	*Author.*				
4	0	10	Lt. Perc. to Hand Mov.	0 0 0	4 0 3	0 0 7
3	4	12	Hand Mov. to $\frac{1}{60}$	1 3 4	2 0 5	0 1 3
2	1	21	$\frac{1}{60}$ to $\frac{6}{60}$	2 0 18	0 0 3	0 1 0
2	0	31	$\frac{6}{60}$ to $\frac{6}{18}$	1 0 31	1 0 0	0 0 0
3	0	9	$\frac{6}{18}$ to $\frac{6}{12}$	1 0 9	1 0 0	1 0 0
0	3	11	$\frac{6}{12}$ to $\frac{6}{6}$	0 3 11	0 0 0	0 0 0
8	2	9	$\frac{6}{6}$ to $\frac{6}{6}$	3 0 0	2 2 3	3 0 0
22	10	103		8 6 73	10 2 14	4 2 10

In conclusion the author wishes to thank the entire hospital staff for the use of their cases.

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THE OPTICS OF THE CORNEA.[1]

By GEORGE W. VANDEGRIFT, M.A., M.D., New York.

IN this paper, necessarily brief, no detailed discussion of the optical phenomena displayed by the cornea is possible. I have attempted to deal succinctly with a few of the refractive, reflective, and diffractive manifestations which are not only of theoretic interest, but also are the fundamental facts upon which ophthalmic optics, in so far as the cornea is involved, are based. It may also, by its very superficiality, create an interest in us to delve more deeply into the generalizations upon which our practice proceeds, and which in our every day haste we are apt to neglect.

The cornea is a transparent optical system consisting of two spherical surfaces and three isotropic media; the air, the corneal core, and the aqueous humour. As the radii of curvature of the surfaces are measured in the same direction as the rays of incidence they are positive and the surfaces are convex.

The anterior surface of the cornea may be divided into two portions, the central lying in front of the aperture of the iris, and the peripheral behind which the iris is suspended diaphragmatically. These two areas thus divided by their orientation differ markedly from each other in their optic manifestations. The central area, essential to the visual function, has an approximate width of 4*mm* horizontally and a trifle less vertically, and corresponds fairly with the average pupillary diameter. The central zone rarely exceeds this measurement but the pupillary width frequently varies up to 6*mm* under which exigency the peripheral area produces many optical obstruc-

[1] Read before Section on Ophthalmology, New York Academy of Medicine, March 15, 1920.

tions to distinct vision, such as spherical aberration, increased size and distortion of diffusion circles and of astigmatic focal lines. The central area is the section of a sphere, or if astigmia be present, a combination of curvatures whose principal meridians at right angles to each other are arcs of two circles of constant though unequal radii. The peripheral area, in contra-distinction, being utilized adventitiously only in the visual act, does not require regularity of curvature and is found in the majority of cases to have a rapidly and continuously decreasing flexure from the border of the central area to the corneoscleral margin where it may become a plane surface or slightly concave.

Of the central area only therefore, is a radius of curvature obtainable, and reviewing the results obtained by many experimenters from Helmholtz to the present day we find 7.75*mm* as a fairly constant average. This however does not imply that a longer or shorter radius produces refractive hypermetropia or myopia for the other optical factors so vary, as axial length and refractive indices, that emmetropia, which depends primarily upon a just proportion of all refractive parts, results.

In a series of fifty eyes examined by means of the Javal-Schiotz ophthalmometer I have been able to determine approximately the outline of the central visual zone. Dividing the arc of the ophthalmometer into spaces of 5 degrees each, and having the patient fix in turn each 5-degree line, a point of intersection is found at which the curvature from a constant reading of the entire central area changes suddenly as the peripheral zone is reached. This may be ascertained both horizontally and vertically in a manner similar to that of determining astigmia ordinarily except that the patient instead of fixing his gaze down the telescope fixes on successive marks on the arc. Such determinations are of course liable to error chiefly on account of the difficulty experienced by the patient fixing the same point in space as the arc is rotated.

The average width was found to be about 35 degrees horizontally, and 30 degrees vertically which is slightly larger than found by Erikson and quoted in the *American Encylopedia of Ophthalmology* vol. 13.

In a similar manner the peripheral zone was analyzed and the curvature found not only to change abruptly at the border

of the peripheral zone, but to increasingly change horizontally and vertically as far to the external periphery as the ophthalmometer would furnish distinct readings.

(*a*) In seven cases in which the central zone was non-astigmatic a 0.50 to 1 D of astigmia developed at about 15° which in common parlance was with the rule, that is, a decrease of horizontal curvature.

(*b*) In thirty-one cases of hypermetropic astigmia with the rule an increase of 1 to 2 D developed.

(*c*) In eight cases of myopic astigmia with the rule the astigmia increased 0.50 to 1 D.

(*d*) In one case of hypermetropic astigmia 0.50 D with the rule the astigmia in the peripheral zone became against the rule 2 D, an increase of curvature.

(*e*) In one case of myopic astigmia with the rule 1 D the periphery displayed no astigmia.

(*f*) In two cases of myopic astigmia of 1 to 2 D against the rule one displayed no astigmia and the other 0.50 D of similar astigmia 15.

In all cases, except (e) the change of curvature that began at about 15° from the fixation line persisted increasingly to the periphery.

The refractive power of the central zone of the anterior corneal surface is ascertained easily when the radius of curvature is known, for the refractive index of the corneal core may be accepted as 1.376 as compared to air which is 1. Refractive power is expressed by the formula $\frac{n' - n}{r}$ in which n′ is the index of the corneal substance, n the index of air, and r the radius of curvature. With r having the value of 7.75*mm* the refractive power is 48.5 D. As the refractive indices are constant the refractivity depends upon the radius of curvature which, while having the average 7.75, may vary from 7.2 to 8.6*mm*. In children the radii of curvature of the cornea as well as of the lens are slightly under the average for the adult, but not in proportion to the axial shortening of the globe; so that while the refractive power of the infant's eye is greater than that of the adult it is due largely to the shortness of the optic axis; the shortening of the radii of curvature being counterbalanced by the smaller refractive index of the lens.

The peripheral zone of the anterior corneal surface approximates a parabola in form and the radii continuously vary. This may be demonstrated by the ophthalmometer as the images of the mires, which at the central zone are rectangular, continuously elongate within the peripheral zone. On account of its parabolic form the peripheral zone produces a positive spherical aberration which is prevalent in nearly all the peripheral regions from the external boundaries almost up to the central zone, and under certain exigencies, such as enlargement of the pupil, natural or artificial, and aphakia, interferes with distinct vision. Under normal conditions a slight aberration is corrected by the lens which has an increasing refractivity from the periphery to the center. Spherical aberration, however, is not entirely due to the parabolic form of the peripheral zone, for even if the entire cornea were the section of a sphere, unless aplanatic, aberration effects would occur, for exact punctual imagery is obtainable in a plane mirror only, and approximately under other conditions only when the incident rays are paraxial.

The following case will demonstrate the practical aspects of spherical aberration:

> Mr. X, age 31, has experienced as long as he can remember, without relief, defective vision and asthenopia. Under excessive illumination he develops a marked photopsia attended by muscæ volitantes, which disappear under low illumination.
>
> Right eye, uncorrected vision $\frac{1}{10}$, left eye, uncorrected vision $\frac{10}{100}$. Cycloplegiac and post cycloplegiac correction with O. D. cyl. $-$ 3 axis 30 and O. S. cyl. $-$ 3 axis 155 furnishes $\frac{1}{10}$ and $\frac{1}{10}$ vision. Tension normal.
>
> Retinoscopy displays a marked scissor movement in both eyes in the axis of the defective curvature. All deep ocular parts normal. The corneæ by oblique illumination appear normal, free of scars or of any tendency to keratoconus. A curious phenomenon, however, occurs with the ophthalmometer. With the mires properly adjusted for the astigmatic reading only a central portion of the combined images appears undistorted while the peripheral parts are markedly elongated.
>
> The symptoms similar to those produced by conical cornea undoubtedly are due to the fact that the central visual area of each cornea is abnormally small, approximately *2mm*, while the average pupillary width is *5mm*, allowing severe

aberration effects to occur under high illumination. The vision was finally tested, with the correcting cylinders worn, through a *2mm* aperture, and that the above reasoning was accurate was proven by the fact that $\frac{1}{1}\frac{1}{1}$ vision was accomplished in each eye. The same result was obtained when the pupils were contracted with pilocarpine.

Data concerning the posterior surface of the cornea are difficult to obtain by experimentation as the ophthalmometer upon which we rely largely for determinations measures the anterior surface alone. It is known, however, to be more nearly the section of a sphere and to have an average radius of approximately 6.8*mm*, which, the refractive index of the aqueous being 1.336, gives a refractive power of − 5.8 D. As the index of refraction of the corneal core is greater than air the anterior surface is refractively convergent, but as the index of the aqueous is less than that of the corneal medium the posterior surface is refractively divergent. The posterior surface therefore reduces slightly the refractive power of the anterior surface.

In calculating the entire corneal refractivity the reduced interval between the two surfaces adds an inappreciable quantity (0.1 D.) to the sum of the two surfaces. The corneal core is of interest optically in the study of diffractive phenomena. This heterogeneous refractive medium may produce halos about the sources of illumination in a manner similar to those produced by tears and discharges in the conjunctival sac. The corneal epithelial and endothelial cells and the cells in the lymph channels obstruct the light rays as they return from the retina, and by unequal diffraction of the light constituents produce spectral halos and rings particularly when the sources of illumination are projected against dark backgrounds. Similarly in glaucoma the penetration of fluids into the posterior corneal layers produces by diffraction the rings and halos pathognomonic of the disease.

When to the corneal refractivity we combine that of the lens, which in an emmetropic eye of *24mm* axial length, under complete accommodative relaxation, is + 19.110 D., computed by means of Helmholtz's constants, we find the entire refractivity of the eye to be + 58.64 D. The anterior and posterior principal focal lengths from the anterior corneal vertex are

-15.707 and $+ 24.387mm$. By these figures an emmetropic eye has its posterior principal focus $0.387mm$ behind the fovea and is hypermetropic approximately 1 D. and must exert some accommodative power to achieve distinct vision at 6 meters.

From formulæ derived from the optical constants the positions of the cardinal points are obtainable, upon which depend calculations respecting diffusion circles, size of retinal images, and other important ocular manifestations too numerous to mention here. A few brief words may be inserted concerning the effects upon these points produced by the process of accommodation. By this act their positions are altered along the optic axis; the focal lengths are shortened, the refractive power increased, and the nodal and principal points drawn nearer the posterior surface of the cornea. If the near point is approximately $10cm$ from the corneal vertex the focal lengths are reduced to $+ 14$ and $- 18mm$ and the refractivity increased to 70 D. The principal points however for practical purposes are unchanged for they are moved by this maximum effort of accommodation less than $0.5mm$. The nodal points are changed more materially as they are much farther from the corneal vertex than the principal points. The principal points, therefore, are the most practical points of reference for the determination of the far and near points, and for the measurement and comparison of retinal images. If we consider the eye reduced to the schematic eye of Donders, consisting of a spherical surface of $\frac{4}{3}$ refractive index and $5mm$ radius, the two principal points coincide and lie at the vertex of this simplified cornea, and the primary focal point from this is $15mm$. The principal point being independent of the accommodative act may be used as the vertex of the visual angle which becomes the measure of the size of retinal images, so that objects having the same apparent size at the principal point have the same sized retinal images irrespective of the accommodative act. But when the anterior focal point is taken as the vertex of the visual angle the retinal image is proportional to this angle for a given value of refractive power. But as the focal angle is independent, approximately, of the ametropia it serves as a comparison of the apparent size of objects perceived by eyes in various phases of refractivity or accommodation.

A discussion of corneal refraction would not be complete

without a mention at least of astigmia. An astigmatic cornea may be considered a pair of crossed cylinders or a sphero-cylinder in which the curvatures are of varying refractivity in different meridians. The normal sections of a sphere are great circles of a sphere and of equal curvature. Their curvatures, however, through a point on the surface may vary from section to section, and if a plane containing the normal is revolved around this normal as an axis one arc will have the greatest and another, at 90°, the least curvature. The sections of greatest and least curvature are always in two perpendicular planes which are the principal sections of the surface at a particular point. The principal meridians of the astigmatic cornea are therefore usually at right angles to each other and may occupy any positions on the circumference of a circle mutually perpendicular. A fundamental law of such mutually perpendicular sections is that the algebraic sum of the refractive powers is constant and equal to the maximum refractivity. A narrow homocentric bundle of incident rays refracted by an astigmatic cornea, represented by a pair of crossed cylinders or a sphero-cylinder, is rendered heterocentric by the unequal curvatures, and punctual imagery is impossible. If the pupillary aperture is small, centered, and at right angles to the optic axis the transverse sections of each astigmatic bundle will be an ellipse, which becoming narrower collapses finally into its major axis. Each of these is the rendezvous of the unequally refracted bundles of light rays, the image line or the focal line of the cylindrical surface whose meridian of curvature is at right angles to it. The distance between the focal lines, the interval of Sturm, is the astigmatic measure. Somewhere between these focal lines, where the refracted rays are equally convergent and divergent, a circle of least confusion is formed.

In common parlance astigmia is frequently denoted by the terms "with" and "against the rule," and by "direct" and "inverse," which are both confusing and inaccurate in the light of freshly accumulated statistics. More accurate and scientific it is to specify an astigmatic error simply by its nature, myopic or hypermetropic, and by the axis of the defective curvature.

The form of the retinal image produced by an astigmatic cornea, whether the defective curvature is horizontal, vertical,

or oblique, is not difficult to construct by placing an imaginary retina at various points on the optic axis. These retinal images are distinctly different from those formed by axial ametropia for in the latter the matter is one of size and definition, while in the former it is one of angular distortion, for the image formed by the defective curvature is larger (hypermetropic astigmia) or smaller (myopic astigmia) than that formed by the opposite curvature. Nor does the wearing of the proper correcting cylinder remove the difficulty fully, for when placed at the anterior focal point a cylinder lens defines the image, but does not affect its size, a result that can be attained only by placing the lens in direct contact with the cornea. It is for this reason that even after proper correction with spectacles patients may continue to complain of image distortion or more rarely of monocular diplopia symptoms, which, however, usually are transient as the astigmatic error is slight compared with the entire ocular refractivity.

When astigmia is considered binocularly the law of corresponding retinal points intrudes for if the astigmia is asymmetric and particularly asymmetrically oblique the images fall upon dissimilar retinal parts, for each image is displaced toward the meridian of greatest curvature. Under such an exigency binocular diplopia may result unless correcting cylinders are worn. Without such correction diplopia must be overcome by sectional ciliary action, which is doubtful, or by exertion of the fusion power through contraction of the oblique muscles rotating the eyes about the anterior-posterior axis, maintaining binocular single vision.

The reflective powers of the cornea are intimately associated with its refractive functions, and are of interest in the formation of the Purkinje-Sanson reflexes, the mathematical calculations of which are of fundamental importance in determining the curvatures and the positions of the refracting surfaces. The reflex from the anterior corneal surface occupies the apex of the visual fixation line so that perimetrically or by means of the ophthalmophakometer the value of the angle α is determined, as well as the positions and centering of the internal surfaces, when combined with calculations concerning the lenticular reflexes. The posterior corneal reflex is produced by refraction by the anterior surface and reflection by the posterior

and with the unaided eye is observed with great difficulty, for the loss here by reflection is no more than 0.02% of the principal bundle of rays while that from the anterior surface is approximately 2.5%. The two corneal reflexes occupy the pupillary plane with the posterior lenticular reflex, while the anterior lenticular reflex, produced by an equivalent thin convex mirror of approximately 15*mm* radius with its vertex 3*mm* from the anterior corneal surface, lies approximately 7*mm* behind the pupillary plane.

Closely connected with these calculations are those concerned with the pupillary image. When we illuminate an eye we see an apparent pupil and iris, the aerial virtual image belonging to the incident rays. This apparent pupil varies in individuals and in the same individual at different ages. The plane of the apparent iris is 0.6*mm* in front of the real plane and the pupillary width is apparently enlarged. By the same refractive process the posterior surface of the cornea is apparently advanced so that deposits that may be upon Descemet's membrane appear by oblique illumination to be in immediate contact with the anterior surface.

A CASE OF RECURRENT CHORIORETINITIS BROUGHT ABOUT BY A SERIES OF DIFFERENT ETIOLOGICAL FACTORS.[1]

By Prof. C. E. FINLAY, Havana, Cuba.

THE clinical history of the case which has given occasion to this paper is as follows:

CLINICAL HISTORY.

W. K., æt. 53, consulted me for the first time on July 24, 1912. He complained of loss of sight, which had gradually developed within the preceding three years, and on being questioned, gave a history of abuse of tobacco, which he had reduced for some short time prior to his visit. I found his eye grounds normal, the vision in each eye being $\frac{8}{10}$, with central scotomata for red and green with normal field limits. I suppressed tobacco entirely and prescribed KI and strychnia internally. By August 17th his vision had improved to $\frac{10}{10}$.

The patient consulted me again on January 7, 1914, on account of a relapse of his visual trouble, which he attributed to a partial return to his old bad habit. I could not discover any lesion in his fundi. His vision: $\frac{8}{10}$; he could not perceive green and presented central scotomata for red. I instituted the same treatment that had been efficient before, a slight improvement resulting.

On May 6, 1914, he returned with another relapse, occurring this time under faithful observance of tobacco abstinence. His fundi showed some dilatation of the retinal veins, some blurring of disk margins, and some loss of transparency at the maculæ. V. O. D.: $\frac{8}{10}$, V. O. S.: $\frac{20}{200}$. Some days later, V. O. D.: $\frac{8}{10}$, V. O. S. : $\frac{8}{200}$. Under a strict enforcement of the treatment which had previously been so effective a slight improvement was obtained but not to the same extent as before. On June 25, 1914, V. O. D. : $\frac{8}{10}$, V. O. S. : $\frac{20}{200}$; the

[1] Read before the Sociedad de Estudios Clinicos of Havana on February 24, 1920.

right eye showing a central scotoma for red and green, the left eye no perception of red, green being only perceived in the extreme temporal field. Tn. in both eyes.

I took advantage of the opportunity of the patient going to Philadelphia to refer him to Dr. de Schweinitz. He had him enter the hospital of the University of Pennsylvania, where his whole system was overhauled, the only defect discovered, outside his eyes, was a dilatation of his transverse colon to twice its normal size. In his eyes he found degenerative changes at the maculæ. He diagnosed a central chorioretinitis brought about by an autointoxication from fermentation of digestive products favored by intestinal stasis in the colon. The patient was given a plan of treatment directed against this. I saw him in April, 1915, on account of a change of glasses. Vision in each eye was $\frac{2}{10}$, the macular lesions being scarcely noticeable.

I lost sight of the patient till November, 1918. He told me his eyes had kept in pretty good shape till the preceding fall, when during a visit to New York, visual troubles, similar to his old ones had developed. He had consulted Dr. Cutler, of that city, who diagnosed a central chorioretinitis and in an X-ray investigation which he ordered an apical abscess was discovered in one of his teeth; this was extracted and an autogenous vaccine prepared and employed. A rapid improvement followed, his sight improving within a few days from $\frac{2}{10}$ to $\frac{7}{10}$. A relapse had occurred a few days prior to his visit to me. As his remaining teeth were sound and he no longer smoked, I had an X-ray taken of his intestines, which revealed great dilatation and sagging of transverse colon. His fundi showed degenerative changes at the maculæ; his vision: $\frac{4}{10}$. I had him use a belt to raise his fallen colon, made him regulate his diet, and in a couple of weeks his vision had improved to $\frac{7}{10}$ plus.

OBSERVATIONS.

The unique feature of this case is the successive production of the same ocular lesion by a multiplicity of causes; in the first two attacks by an intoxication with tobacco, in the next by an intoxication of intestinal origin, in the fourth by septic absorption from an apical tooth abscess, and in the last attack again by an intestinal autointoxication. The possibility of all the outbreaks being brought about by this latter cause can be excluded by the rapidity with which the visual disturbances responded to treatment not taking this into account in the other instances. To my mind this can only be explained by

some local lesion affecting the macular regions of our patient affecting their metabolism or making their nutrition difficult under the slightest variation from normal of its vascular supply and especially susceptible to certain toxins or poisons, the disturbances being at first purely functional. Arteriosclerosis is most probably at the bottom of the whole process.

The case is also of interest in connection with the etiological factor in each individual outbreak, lending itself in each instance to a review of the status of our knowledge regarding them and to considerations as to how much light it may throw on some disputed points.

In connection with the tobacco attacks, we find the case as pronouncedly favoring the theory of the primary lesion in tobacco amblyopia being in the ganglion cells of the macula. Prior to 1882, ophthalmological literature is rich in considerations and descriptions of the clinical aspect of this affection, some authors advancing different hypotheses as to its pathogenesis. After Samelsohn's (1) first anatomical findings, which were subsequently confirmed by Nettleship (2) and Vossius (3) the disease was ascribed to an interstitial retrobulbar neuritis of the papillomacular bundle. The lesion was later thoroughly worked out by Uhthoff (4), who traced the bundle from the macula to the basal ganglia. These investigations seemed conclusive but the latest researches point to the lesions found in the optic nerve being secondary to changes in the ganglion cells of the macula (5). Our case gives clinical support to this latter view, it not being surprising that in the first attacks no ophthalmoscopic changes were discernible at the maculæ as possibly no actual lesion then existed, the trouble being purely functional or they may have been so little pronounced as to have escaped detection—the fact that they showed up later makes one or other of these possibilities probable. In cases of tobacco or alcohol amblyopia with a marked atrophy of the temporal quadrant of the disk (papillomacular bundle) with no visible changes at the macula it is perhaps possible to admit that the lesion be essentially a retrobulbar neuritis interstitial in character, but in cases like this where no such atrophy was apparent even after the appearance of ophthalmoscopic signs at the macula, this hypothesis is not tenable.

The production of a central retinochoroiditis as a result of an intestinal autointoxication agrees with the researches of de Schweinitz (6) and other observers and one of the attacks which he had the opportunity to observe was attributed to this origin after an exhaustive investigation at the Pennsylvania University Hospital. The results of the dietetic treatment instituted in the first attack and of the mechanical means employed to prevent intestinal stasis in the last attack I saw him in, bear this out. I did not have the opportunity to try with this patient the treatment recommended in such cases by J. G. Dwyer (7) but if I meet with a similar case I propose to give it a trial.

The production of a focal eye lesion by septic absorption from an apical tooth abscess in the attack treated by Dr. Cutler, of New York, is in line with the numerous cases reported since Hunter, Lang, Ulrich, and others called attention to this etiological factor in eye diseases of obscure origin (8) and is confirmed by the rapid improvement following the extraction of the guilty tooth.

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OCULAR ORIENTATION: A FUNCTION OF THE EYES. ITS IMPORTANCE IN OPHTHALMOLOGY; ITS DEPENDENCE ON THE MUSCULAR SYSTEM.

By E. H. HAZEN, M.D., Oakland, California.

EMERITUS PROFESSOR OF OPHTHALMOLOGY, DRAKE UNIVERSITY, DES MOINES, IOWA.

ORIENTATION is a word that has come down to us from ancient lore, through the churches later, and was soon applied to an instinct of animals that could find their way home after much wandering, and is now taken up (first I think) by Fuchs, to describe the function of binocular vision in man.

It is upon this function we depend for the guidance of every movement we make, from carrying a fork to our mouths, to the aviation of the sky. It is responsible for our efficiency in every calling we follow, whether for pleasure or in earning our livelihood. It is our pilot in our sojourn, from the time we leave our mother's apron string, until Father Time cuts the swath that gathers us in.

It seems singular that we have not taken up this important function as an entity in ophthalmology, and sought out the attributes belonging to it in health and disease, and discovered tests to learn its abstruse office, and detect its many disorders, *and put it in the fore rank of other branches with which the oculist has to deal.* For if I am not much mistaken, in the controlling apparatus of this function, is seated the octopus of human ills we call asthenopia.

We need a clearing up of the nomenclature of this function-orientation. *Orientation* covers the expression of this function, but there are divisions in it of faculties that are distinctive.

Projection is looking, regarding, it is the direction of casting an eye, and ought not to be confused with other faculties of orientation. This has to deal with the fovea, the axis, the nodal point, and the accommodation. It is a congenital faculty, and is concerned in obtaining a clear picture on the retina, of the object contemplated. The science of refraction administers to its defects.

The faculty that gives us dimension, situation, bearings, depth, relation, distance, contour, solidity, form we may call ***perspective. This faculty is an acquired one; gotten by experience.*** "The maintenance of correct binocular vision is the necessity which dominates the ocular muscles" (Noyes). The important function of ***fusion*** is here a prominent feature.

On this faculty depends efficiency in action, and safety from accident. In normal eyes there is a quickness and facility of movement exceeding the muscular movement of all the other muscles of the body, and upon this system of muscles the brain is furnished information for judgment of action. "The image falling upon the periphery of the retina gives us warning signal to cast our eyes upon the object which excites the image. It is moving objects that are most sure to attract our attention, for the peripheral portions of the retina have a higher degree of sensibility for the perception of movement" (Fuchs). There is no other faculty in us that gives more pleasure and enjoyment of life; greater buoyancy and vivacity of spirits and manner, larger confidence in the initiative. One with this function normal gets credit for sagacity, acumen, common sense, discrimination. This is because acute observation depends upon freedom of action of the eyes. There is built upon this easily acquired natural trait, confidence and courage and self-efficiency.

From an experience of twenty-five years in treating the muscular system for asthenopia, with view of getting free action of these muscles, with unexpected success, and of late concluding that in doing so, I have been administering to the disordered function of orientation, I believe I have thus allocated these several faculties that have been so abstruse in ophthalmology.

The repugnance among those who administer to eyestrain to listen to the detailing of symptoms associated with eyestrain,

may be attributed to the want of appreciation, on the part of the oculist, of the *function of perspective*, which is disordered, and which the patient is attempting to describe, though, himself, unaware of its entity; also to the habit of the practitioner to attribute the causes of the phenomena of all eyestrain to errors of refraction (mechanical) and correcting the slightest defect at a venture. If his attention could be directed to an apparatus having control of as broad and important a function as orientation, he could better appreciate and properly place the symptoms described, and seek a pathological method for its solution.

We have been a long time finding the elements of the function of sight, and properly allocating them; we have applied a deal of the sciences of algebra, geometry, physics, and surgery to the mechanics of the question, but have neglected the physiological side of the problem. For fifty years we have practiced Professor Donders's system of *Accommodation and Refraction* and what we have learned of the complication of disorders of the muscular apparatus in the function of these organs, we have depended upon correcting through the focal apparatus and tenotomy of the muscles.

The art of glass fitting is the administering to the function of *projection*, and goes no further. Besides to seeing well, which glasses afford, there is more frequent application for pain or eyestrain, and as one of Donders's tenets is "that hypermetropia is at the bottom of it," the adjustment of glasses has been almost the sole means of caring for asthenopia.

There is no doubt that the apparatus of motor muscles of the eye, is the mechanism and controlling power of orientation, and has an independence of the focal function, connected with it only by nerves. "In other parts of the body the muscles are constructed for strength rather than speed, but with the eyeball everything is adapted for speed" (Maddox).

It is subject to disorders, and needs as much, if not more, repair than the focal part. I have treated the muscles by gymnastics with the idea of strengthening the muscles, but of late years believed that in the process which carried this out *I was getting rid of obstruction, probably débris of hyperemia or inflammation*. The hyperemia of the retina would clear up, the conjunctiva and the sclera would very soon get rid of its

vascularity, even iritis would be dissipated. I have concluded from these experiences that the pathology of asthenopia, is the deposit of this débris, and in the exercise of the muscles I have pumped it into the circulation, leaving freedom of action and cure of asthenopia. I have so announced this in a paper written last June, which appeared in the January number ARCHIVES OF OPHTHALMOLOGY. In this article I give the process in detail.

From the fact that this irritation, from whatever nature it may be, can disturb other nearby nerves by reflex, gives us the variety of symptoms that have been traced to asthenopia as a cause. We have seen many who had difficulties of vision, but had no pain, but would refrain from using their eyes in some kinds of work. There are people that seem to have nerves of wire and very little sensibility, and yet have deflections or imbalances.

Then again, it is very true that many people pass through life without having trouble with their eyes, or at least do not complain, but among those who claim good eyes, there are many who are deficient in the faculty of perspective, and who are unable to overcome a 10° prism by the laterals; although they do not have pain many are slow in movement and cannot fix readily.

When this function is better understood, and the value of it properly appreciated, the tests for eye phenomena that have been recorded, will have to be revised, and based upon experiments made on those of proved healthy eyes. It is found that Helmholtz must have had some defect of his eyes, as some of his observations are not verified.

If there is truth in the assertion of Tscherning that we measure distance and relation of objects by the sense of the amount of innervation expended in performing the act of looking, then when these muscles have obstructions in movement and require expenditure of innervation, more at one time than another for the same movement, we have no criterion to base a measurement in orientation in eyes that have asthenopia.

We have also to deal in this function of orientation with light sense, color sense, and form sense, the faculties of accommodation, convergence, and direction and the conjugation of the two eyes to act as one organ.

Perspective, or that use of the term in the fine arts: the perception of space, form, depth, color, light, and shade, is usually thought to be an acquisition of the æsthetic and cultivated by the intellectual, a requisition of the cultured. There has been no thought of its being a leading factor in the skilled artisan and practiced every day by the laborer in the use even of the pick; or that it has an intimate relation to every one's safety in every day perambulations. Nor is it taken up by the ophthalmologist as a function important to be preserved in good order. Neither are the deficiences of perspective or orientation described by patients as such, but they complain of difficulties experienced in vision which should be assigned by the oculist to anomalies of the muscles—the apparatus which functions perspective.

Let us now convert this significant æsthetic attribute of the beautiful, into practical current everyday conduct. One who has lost the sight of one eye is not only handicapped in the social realm, but he is incapable to perform many of the requirements in manual labor and other industrial pursuits, and is continually meeting with accidents, though he learns caution and goes slow. He cannot drive nails and cannot compete in mechanics, and should not be about machinery.

A ranchman told me of a man in his employ, who when harrowing in his orchard, was continually barking his trees, and as he regarded him as a willing and intelligent workman, it puzzled him to know why he should be so careless. When I told him the fact that a man with one eye could not measure distance, he accepted the explanation and kept his one-eyed man on other work. The man changed his employer, but soon left his second place, and now is selling stoves. I often meet one-eyed collectors. A one-eyed man should always carry a cane. No man with but one eye should be a motorman or run any kind of vehicle, much less try aviation.

But a man with one eye is not troubled with asthenopia, for asthenopia comes from the defects of binocular vision. There are cases reported where the surgeon had given up in despair his ability to relieve his patient with two eyes, of the terrible distress of binocular vision, and he proposed enucleation of a good eye. It is an actual fact that there are several cases in which this has been done.

One characteristic of asthenopia is its periodicity, and another that often these cases can freely enjoy themselves, perhaps for hours, and not feel any inconvenience, and retire feeling well, but before morning will suffer severely, and are often confined to their rooms one or two days. This is pathognomonic of its reflex nature; sea sickness is another example —a reflex upon the pneumogastric. Another prominent symptom is a dread of motion. They cannot endure the "movies"; the quick fixation required, is wanting.

We all know of the almost universality of nervous symptoms in American people. In this country there are more hours given to reading than in any other nation; the masses are better educated and employments are so subdivided i to special duties that very few muscles are called into the tasks, and in nine-tenths of them the eyes are used severely; then this apportionment of work calls into action so few other muscles, that the muscular system is not evenly built up or kept vigorous, and the power of resistance is weakened, and nervous symptoms are developed.

Again: The symptoms of disordered eye muscles, often have such subtile and mysterious phenomena, that they are difficult to depict or to understand whether they are belonging to the physical, mental, or moral nature, or simply are idiosyncracies of disposition. The collection made by Dr. Geo. M. Gould of expressions of men, who undoubtedly had asthenopia, and who are used to literary expressions of the most graphic kind, I regard as a contribution to the subject I am trying to elucidate. The doctor's mistake is in attributing the phenomena to the need of proper lenses. We know something of the percentage of people who have some reason to complain of their eyes from the amount of glasses worn. There is an army of men making a living out of it. More than half of these applicants for relief, at least, have the trouble wholly in the motor muscles, and who discard their glasses within two years as useless.

I believe that one half of the community have asthenopia, and two thirds of these have disorders of the apparatus of orientation. The theory I have advanced as to the pathology of asthenopia, fits well into the phenomena, both as to the course of the ailment, and a working basis for relieving it.

The subjects of this disease are found in every occupation, and thousands are driven out of remunerative employment to take others "for want of adaptability." Some are aware of their affection and some are not. A young preacher became a book agent, because he could not study. The treatment of his eye muscles, fifteen days, one treatment a day, enabled him to go back to his chosen work. A dentist played the trombone in an orchestra at night; one evening everything turned black before him, and he tumbled off his chair. He was put to bed for biliousness. The treatment of his eye muscles cured him of asthenopia, though he had tried glasses, and he is now at his former work. An electric worker, who did inside work, was no longer able to stand on a ladder. His physician doctored him for indigestion for three months and then told him to consult me for his eyes. He was surprised and declared he had good eyes, but he came: Vision $\frac{20}{XX}$, orthophoria, but weak duction. In less than ten days he went to work, and remained cured for two years, when I lost sight of him. I have the records of scores of teachers who gave up their work, and could only do housework; also a number of students who broke down in college. One miniature painter who had learned her art in Europe, and opened a studio in New York City, broke down, closed her studio, and came home to Iowa and for two years could do no palette work. In two weeks I cured her and she opened a studio in Chicago, and took the prize for miniature painting in Philadelphia. A poor woman over fifty years of age could only do kitchen work; gave up reading for years, could not read the papers. Treatment of a very few days enabled her to read. During treatment she asked, if I cured rheumatism with that instrument. She had had cramps in her toes and folding of her fingers, but since treatment for her eye muscles, she was entirely free from the ailment. A ranchman was troubled with intolerance of light; wore black glasses out-of-doors. In forty treatments, all at once nearly at the end of the treatment, he found he could go in the light without his glasses, and throw back his hat without discomfort in the sunshine of Oregon. He remained well when last heard from six years after. In one case, treated for weak muscles and lid congestion, attained to standard in twelve treatments. One year after she reported that a goiter she had had for years,

had disappeared. Car sickness of twenty years' duration, after being fitted with compound glasses by one of the best reputation in the country, was entirely cured, and would not wear her glasses afterward. For three months I had her under observation and there was no return of trouble.

I have cured three cases of chorea, and I believe one of locomotor ataxia, though an osteopath, who I found was treating her at the same time, claimed it. Two cases of enuresis were relieved.

I might truly enlarge on the prevalence of asthenopia, and its remote symptoms to such an extent that it might excite ridicule, but in my experience of practicing and teaching this subject, I have repeatedly had strange and seemingly foreign symptoms related to me in muscular treatment that were relieved, that would compel me to believe they belonged to this yet obscure and neglected function; symptoms that were responded to by physicians with a grim smile and "thrown over their shoulder" and which produced from them a moral lecture on self-control, etc.

The most persistent interpretation of symptoms by general practitioners (especially if the patients had frequently changed their glasses), was that some remote cause produced them, and a six-months' rest in sanatoriums or a course of treatment was instituted. We seem never to get rid of the idea that neurasthenia produces eyestrain, but I am convinced that the most frequent is that eye trouble produces neurasthenia.

There is one expression of this disease which I can only name desperation; it often comes in waves which overwhelm the patient, and he cannot sit still, but dashes away and walks it off. This feeling of desperation has often been described to me, and leads me to believe that in some suicides and other crimes there might be some explanation for them in asthenopia.

Knowing, or at least believing, that car- and seasickness also belong to a disorder of this particular function, showing a reflex on the pneumogastric nerve, I have often thought that "heart failure," a mysterious and senseless diagnosis, might have some connection with asthenopia.

My excuse for the details of symptoms is for the purpose of showing the connection of these widespread nervous conditions with this function orientation. This function has such great

utility in the walks of life, and has such a diversity of manifestations of its ailments, that it is of first importance in ophthalmology.

I base my thought of repair on this apparatus of orientation, on disciplining the muscles that they may be free from spasm and work with perfect mobility and quickness of action.

I have long since come to the conclusion, that the muscles of the eyes are constructed and attached to the ball in accordance with the environment of human uses of that organ, and have long since quit whittling them. They have a wonderful fitness and adaptability to circumstances, and in time can adapt themselves to new requirements. Undoubtedly the condition of parallelism is best fitted from which to start on duty, and to which to return when it is performed. This is the point of rest when the eyes are open; this, of course, the point to which we should aspire to bring them, and give them as much ease of action as well as swiftness to call to duty.

I would not be understood as ignoring refraction. Many a pair of eyes have been relieved by correcting ametropia, but the fitting of all eyes that hurt with a pair of glasses, is a terrible exaggeration of the art. The attention to the apparatus which has in charge the orientation outranks it in importance.

AN ANALOGY.

The American watch is a beautiful and a marvelously perfect mechanism, and is now so common an instrument in the economy of human affairs that it has become a matter of course that it should, and really does, keep good time. But in the regular adjustment of business affairs it is a small part, for instance, in the keeping of an appointment. It does nothing towards getting the man to the agreed location; the motor appliance of transportation is independent of his watch; it fulfills an important point, but the orientation must be accomplished by another process. So I may say with some apology, that focus of the eyes in the function of vision has but the same relation as the timepiece has in the active transactions of everyday business.

The faculty of perspective has an almost independent rela-

tion; the universal (pretty nearly so) usage of prescribing glasses to this apparatus of perspective for eyestrain, and ignoring the delicate and most important part of the apparatus of orientation, is a travesty on ophthalmology.

Suppose in the morning arraignment at the Police Justice Court a "drunk" was lectured thus: "Mr. Jones I am convinced that your plight which caused you to be 'run in,' was owing to the fact you had no watch. If you had had, you would not have so blundered by your inability to orientate and get so far from home. You were no doubt, surprised to find yourself in jail this morning; your sense of location was destroyed, and you were in great danger of losing your life. I advise you to procure two watches, and that you have them repaired here, every three months."

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. CONRAD BERENS, Jr., Secretary.

MEETING OF FEBRUARY 16, 1920. DR. A. E. DAVIS, CHAIRMAN.

Dr. Daniel H. Wiesner presented a patient, a child of 9 years, with bilateral **disseminate choroiditis** and no central vision in the left eye. Both the Wassermann and the tuberculin tests were positive.

The subject of the evening was **wood-alcohol poisoning.**

(a) **Clinical aspects**, with special reference to the eyes and Committee report.

Dr. Coleman W. Cutler, after speaking of the important work done in this country by Wood and Buller, Holden, Allport, the Committee for the Prevention of Blindness, and others, described the general and ocular symptoms of wood-alcohol poisoning, emphasizing the two following points: First, unless the case is seen very early and the diagnosis made and reported at once, there is little chance of obtaining the evidence required to convict those who have sold the poison. The law has frequently failed because of delay in obtaining evidence. Second, cases of optic atrophy come to our clinics not infrequently in which the cause is undiscovered.

Chronic changes occuring when wood alcohol is used as a solvent for shellac in hat stiffening are chronic conjunctivitis, moderately congested or atrophic optic nerves with vision reduced to one-half. Acquired tolerance has been noted.

The Committee appointed by the Section of Ophthalmology reports that it has conferred with the Committee on Public Health of the Academy of Medicine; that measures were taken

at once for a conference with the President of the Wood Products Company; that the attention of the Board of Health was called to the fact that wood-alcohol poisoning was not reportable and that urgent measures were needed to enforce existing laws.

As has been stated, the name methanol has been substituted for wood alcohol and the strength of the denaturant reduced from ten per cent. to two per cent., thereby rendering denatured alcohol innocuous. The meeting to-night is a final result of the work of this Committee.

(b) The **pathology and pathological chemistry of wood-alcohol poisoning** by Dr. CHARLES NORRIS.

Attention was called to the prominent part which ophthalmologists, especially those of our country, have played in arousing the community to the dangers of this form of alcohol. He entered into a discussion of the difficulty in the determination of the cause of death in many of the cases which come under the jurisdiction of the Chief Medical Examiner's office.

Attention was called to the slight, indefinite and unsatisfactory results obtained in the microscopic examination of the sections of the nerves, especially of the optic nerves, in the cases which have come under his observation, the reason being that the cases were rapidly fatal cases of poisoning, in which one does not expect to find the lesions described by Birch-Hirschfeld, Holden, and others after experimental poisoning in animals.

The necessity of chemical examination of the viscera in suspected cases of poisoning was emphasized.

A statistical table was given of the number of cases of methyl alcohol poisoning which occurred during the years 1919 and 1918 in New York City.

(c) Some **chemical aspects of the wood-alcohol problem.** Professor CHARLES BASKERVILLE described the manufacture of wood alcohol and its legitimate uses. He favors the elimination of the name alcohol as of great importance removing as it does the symbol or idea of the intoxicant which the qualifying "wood" or even the poison label has not always accomplished.

(d) **Legal aspects,** Professor JOSEPH P. CHAMBERLAIN. The problem of restricting the illegitimate uses of wood alcohol, is one of regulation and belongs to the nation rather than the

State. National regulation can best be effected by taxation. This result could be achieved by taxing wood alcohol and by allowing withdrawal of the alcohol tax free for shipment to manufacturers, the tax to apply the instant the product was sold to any but a registered manufacturer.

DISCUSSION: Dr. WARD A. HOLDEN: The warning that the public has received, together with the present status of the wood-alcohol industry, seem to guarantee us against any great amount of wood-alcohol poisoning in the immediate future.

The favorable features as regards the industry are three in number:

1. The refiners of wood alcohol have changed its name and now designate all grades as METHANOL.

2. The restriction in forestry operations has cut down the supply of wood alcohol, while the demands for wood-alcohol products in agriculture, moving-picture films, dye industries and the like, have been greatly increased, both here and abroad, thus raising the price to $1.80 a gallon.

The legitimate industries in the future will take the entire output, leaving none to be scattered in small lots, as in the past, in pharmacies and paint shops.

Denatured grain alcohol which costs but 60 cents a gallon, one third the present price of wood alcohol, will soon entirely take the place of wood alcohol for use by the general public.

3. Because of the scarcity of wood alcohol the Federal Government now requires in the production of denatured grain alcohol the addition of but 2% of wood alcohol as compared with the 10% required before January 1, 1920. And it is likely that, following the lead of European countries, the Government will soon permit the use of a lower grade of refined wood alcohol which is less toxic and also more nauseating than the grade now required. Hence the danger from denatured alcohol is greatly lessened.

In regard to legislation, we have gone slowly, knowing that any bill that hurts the refiner will be blocked by him, and any bill that hurts the other industries will be blocked by them. Further Federal legislation may prove to be necessary, but it should be very carefully considered from every point of view, and no Federal supervision should be urged that will appreciably discourage industries and increase the cost of living,

which Federal supervision invariably does, unless we can be quite sure that it is not needless, and that it will diminish materially the prevalence of wood-alcohol poisoning in the future.

Dr. H. W. WOOTTON stated that he had recently been able to examine the fundi in two cases of methyl alcohol poisoning shortly after the onset of symptoms. In both, the retinal veins were somewhat dilated, but there was no marked papillation. The disks were pale and their outlines hazy, but there was no distinct elevation. Perception of light was absent and the pupils widely dilated. Both recovered sufficient vision to enable them to go about, and later the disks became very pale. He did not think that a further improvement was possible. In fact, one of the cases after three months showed a renewal of decrease in vision. The fields were contracted.

Dr. CRIGLER said that a case recently seen by him in association with Dr. Wootton emphasized the importance of a change in the name of this deadly poison. A Russian Jew age 30 had deliberately consumed the contents of three three-ounce bottles of wood alcohol—taking only a small quantity at a time—despite the fact that each bottle bore a poison label as required by law, and despite the fact that he intelligently read the labels; he said he thought that he could take a chance by taking only a small quantity at a time; recovering from the effects of the first few drinks with only slight drowsiness and abdominal pain—which he attributed to something that he had eaten and not to the poison—he took several larger drinks in succession. His abdominal pains became so pronounced that he was compelled to seek relief. Returning from the doctor's office he became unconscious, was taken to Bellevue Hospital and remained in a comatose state for three days. When consciousness returned he was totally blind. There was a gradual restoration of his sight so that at the end of six weeks his vision in the right eye was $\frac{20}{70}$ and in the left, $\frac{20}{100}$. His vision to-day, three months from the date of the poisoning, is $\frac{20}{100}$ in the right eye and $\frac{8}{100}$ in the left; the fields are markedly contracted; color perception is almost completely abolished and the nerve heads are white with an atrophic excavation. The arteries especially are considerably contracted in size.

MEETING OF MARCH 15, 1920. DR. A. E. DAVIS, CHAIRMAN

Dr. S. A. AGATSTON presented a patient with **sarcoma of the left orbit,** extending from the antrum.

F. L., male, age 64 years, hospital orderly. Three months ago began complaining of pain in left side of his face. He was treated for facial neuralgia. Two months ago his left antrum was irrigated, but no pus was found. The pain continued more severe up to the present time. Three weeks ago, the patient noticed the protrusion of his left eye and swelling of his face. Since that time the swelling steadily increased. At times there was diplopia. The patient lost twelve pounds in weight during the past three months. His family and previous history are negative.

Present condition: Swelling over the left maxilla; pulsation of his left cheek and left eye, but no bruit; hyperæsthesia of the area supplied by the infraorbital nerve; tenderness on deep pressure over the malar bone; increased respiratory sounds over the left antrum; exophthalmos of about 8*mm*; limitations of ocular movements are only slight; diplopia in the lower field; chemosis of the lower half of the conjunctiva; pulsation of the left fundus synchronous with the pulse; left papillœdema. Vision: O. D. $\frac{20}{20}$, O. S. $\frac{20}{70}$. The fields for colors are contracted. X-ray shows diminished illumination of the left antrum, with diffusion of the outer border suggesting a new growth. Wassermann negative. No evidence of growth elsewhere in the body.

A tentative diagnosis of sarcoma of the antrum, extending into the orbit was made.

Dr. AGATSTON also presented a patient with **dislocation of the lens into the vitreous chamber.**

Patient G. J., was struck in the eye by an unknown object thrown at him. He was brought to Bellevue Hospital (Dr. May's service) with swelling and laceration of the lids as well as subconjunctival hemorrhage. The cornea and anterior chamber were clear. There was no retinal reflex and it was impossible to see the fundus, owing to hemorrhage in the vitreous.

After a few days, examination showed the presence of a tremulous iris, the lens dislocated toward the nasal side and tilted backward, the convex edge seen across the pupillary area;

the vitreous filled with large opacities. The next day, the lens disappeared from the pupillary area, but was found at the bottom of the vitreous chamber. X-ray showed no fracture, or foreign body in the orbit. Vision: hand movements. Fields normal, but no color perception. Tension 18 (Schiotz).

Dr. DANIEL H. WIESNER exhibited a patient with **deep keratitis**, in whom the Wassermann and tuberculin tests were positive. The child has been under observation eighteen months and has had treatment for lues and tuberculosis. Two weeks ago the patient developed bilateral inflammation of the irides with spots on Descemet's membrane. The twin sister gave a negative reaction to the Wassermann test and a tuberculin test was only slightly positive.

DISCUSSION: Dr. H. H. TYSON stated that it was not uncommon to find tuberculosis and lues in patients with interstitial keratitis and that while both may exist, one or the other might be the predominating cause, while in other cases, both may appear to share equally as the causative factor. Judging from the appearance of the cornea in the case under consideration, he was of the opinion that tuberculosis was the predominating cause and suggested continuing the tuberculin treatment and the use of tonics; later anti-luetic treatment if necessary. He advised against the use of potassium iodide in patients suffering from tuberculosis as he was of the opinion that it was more injurious than beneficial.

Dr. A. E. Davis suggested that the mental inferiority of the twin child affected with the keratitis as compared to the well child was only apparent and that the backwardness of the sick child was due solely to the fact that she could not use her eyes at school.

Dr. ALEXANDER DUANE demonstrated what he called the **complementary color test** but what might better be called the extinction test.

This is usually applied with the tangent curtain, and, for examining central scotomata or the blind spot, at a distance of sixty inches; for examining hemianopsia at a distance of thirty inches. Suppose we are examining the left eye. The right eye, covered with either a deep amber or a ruby glass fixes the central spot of the curtain. The left eye is uncovered. A dark blue disk on a carrier is pushed in various directions along the

curtain. This spot is invisible by the right eye, because extinguished by the colored glass. It is visible to the left eye as a rather lighter blue, until it enters the scotoma when it disappears sharply if the scotoma is absolute or becomes dim and dark if the later is relative. The red glass is particularly serviceable in testing the blind spot, because with this any straying from binocular fixation is rendered evident by the fact that the little white central spot used as the point of fixation appears as two spots, one red and the other white. If the patient's attention is called to this fact and he is made to fuse the two images, sharp central fixation with the left eye is maintained and we get no uncertainty about the limits of the scotoma.

Dr. DUANE read a paper on **some theoretical and practical points in refraction work** (appears in this issue.)

Dr. G. W. VANDEGRIFT read the second paper on **the optics of the cornea** (appears in this issue).

DISCUSSION: Dr. ELLICE M. ALGER said that most ophthalmologists assumed that refraction was a finished subject, to which nothing of importance had been added since the time of Donders and Helmholtz. We were rather proud of the statement that it was nearer to an exact science than any other department of medicine.

Theoretically, modern methods made it possible not only to save time but to rely almost entirely on objective examination. Some men even had as their ideal a system under which it was not necessary for the patient to open his mouth. If refraction were an exact science, it naturally *should* follow that any well-trained man could do it, and that all should get practically the same results. If the patient was not comfortable with the resulting prescription he is assured that his examination was made carefully and scientifically and that it was his misfortune if he could not wear his glasses. Moreover, doing this simple, scientific, objective, impersonal thing over and over bored us to death. It was as exciting as examining a series of schematic eyes, and most men looked eagerly to the time when they could shift this drudgery to an assistant.

Practically, however, we all knew that refraction was more than a science; it was an art based on a science, in which the more scientific man was not always by any means the more successful artist. Moreover, practiced as an art, which allows

for judgment and experience and even intuition, it became interesting.

Dr. Alger thought we were paying too much attention to static measurements and not enough to dynamic ones. Our very terms were misleading, for the emmetropic ideal eye was often far from the perfect eye in actual use while the ametropic eye often functioned perfectly.

Accommodation was a very complicated function, which was not to be done voluntarily but only under the imperious desire for distinct vision. Some people could not even think without fatigue and were entirely unequal to even the nervous stimulation of accommodation. Others probably lacked the knack of coördinating perfectly normal muscles, like the athlete who was so muscle-bound that he could not throw a ball.

The elasticity of the lens we recognized as an essential element in focusing, greater in childhood and declining with age, but whether this decline was purely a matter of age or depended partly on factors more or less under our control, like diet or toxemia, we knew very little. It was even possible that the lens, like an India-rubber band preserved its elasticity much longer when actively used. An inelastic lens caused poor vision but did not in itself result in discomfort, which proceeded from the muscular element in accommodation.

The natural instinct for clear vision led us automatically to stimulate the ciliary muscle, not a measured amount, but till we got results. When the most energetic squeezing failed to clarify vision, many people never seemed to learn the futility of the effort but kept straining clear into old age and complaining bitterly of the pain and fatigue.

When we measured accommodation we really measured the change in the lens and had no idea of the amount of energy used in producing the result. As far as actual power goes we had no reason to believe that the ciliary muscle of a healthy adult was essentially weaker than that of an infant. In the one a slight contraction produced a tremendous result while in the other a much more powerful one produced hardly any result at all.

The art of refraction is very largely a study of the muscular compensation for ocular defects, and cannot be done by rule of thumb. If we removed part of the load we removed for the

time being all the patient's disability; he could take care of the rest. It was even possible that we did him more harm than good by removing too much of his burden, by strong glasses, just as the cardiac patient often did much better under graduated exercise than under complete rest.

When compensation had actually broken down it often required the nicest judgment to decide the best proportions of rest and exercise and glasses.

Dr. Alger was sorry to see the growing dependence on cycloplegics; not that they were not often necessary, for they were, but that in many cases the patient's actual needs could be better met without them. They often obscured vital elements in the problem and sometimes seemed to paralyze the initiative of the physician even more than they did the accommodation of the patient. They should be omitted or used according to needs of the individual case.

Dr. Alger thought there were far too many people in the world wearing weak glasses all the time, who would be much happier with no glasses at all, or stronger ones for near work only. Many of us had suffered this in our own persons. We were willing to admit that glasses were a great blessing, but they were also a nuisance. If we put them on a patient it should be only after a study of his individual needs has convinced us that they were really indicated, and not because he had a good nose to hang them on.

Dr. J. HERBERT CLAIBORNE said there were two things that nature seemed to abhor—a blurred image on the retina, and more particularly a distorted one; that the accommodative apparatus was intended primarily for the purpose of focusing divergent rays of light within twenty feet, and that the impulse was so profound, the mind naturally turned to the accommodation as the only means of eliminating a blurred focus on the retina. In the case of spherical hyperopia, this is done by the muscle of accommodation but in hyperopic astigmatism the apparatus was incapable of overcoming the blurred image by reason of the difference of refraction in the two principal meridians, nevertheless, the brain would attempt to overcome the blurred image whenever it occurs. This is the cause of accommodative strain which produces so many symptoms. A distinct image is desired at any cost and the cost of the futile

effort or the successful effort is paid for in the coin of pain or other discomfort.

It is safe to say that seventy-five per cent of our work is refraction, hence the importance of this subject. He used cycloplegics in all children and generally in people up to approximately thirty-five years of age, but he insisted on three examinations before, during, and after the cycloplegic. This was necessary to render a correct judgment. Both science and art were involved in the correction of the refraction of the eye. A knowledge of optics was absolutely necessary and was the basis of all further investigation but the art consisted in applying these principles and many others which are ordinarily not considered a part of science but in reality are. They are character, patience, persistence, knowledge of psychology, charity of heart and soul, loving-kindness and the realization that the patient knows what he wishes, knows when he has it and hasn't it, and has quite as much sense, if not more, than the examining surgeon. He recommended to young men in preparing themselves for the correction of refractive errors, to make a study of anatomy, physiology, mathematics and physiological optics, and then, after much prayer and fasting, to prescribe with fear and trembling. The main object is to satisfy the patient, and make him see well with comfort and ease.

Dr. A. EDWARD DAVIS spoke of his experience with the use of the Souter ophthalmo-phakometer. We are indebted to Dr. Souter for adding one more valuable objective test in estimating and correcting refractive errors. As you are aware, with this instrument we not only measure the corneal astigmatism, but the lenticular astigmatism. Dr. Souter has stated that after measuring many hundreds of cases of refractive errors with his instrument he found that the sum of the corneal and lenticular astigmatism (the posterior lenticular surface is the one chiefly affected in astigmatic errors) conformed with the subjective astigmatism to within .25 D. in the vast majority of cases. His experience in the use of the instrument agrees entirely with that of Dr. Souter. Even before the advent of this instrument, he rarely made use of cyclopegics in refractive work in patients over fourteen or fifteen years of age and since employing it, he finds even less necessity for using cycloplegics as a routine measure in fitting lenses.

Dr. DUANE in closing said that the apparent discrepancy between his statements and Dr. Vandegrift's with regard to the effect of the posterior surface of the cornea on the refraction of the eye was due to the fact that Dr. Vandegrift was considering only the effect exerted on the refraction of the cornea itself while his own paper had regard to the effect produced on the refraction of the whole eye.

Answering Dr. Alger, he said that Hess had assumed that the actual ciliary power was the same in people of nearly all ages, but that, as the lens hardened, more and more of this power became latent because less and less was needed in accommodation. A necessary corollary of this view is that paresis of accommodation must escape notice in people of middle age. But experiments made with homatropine prove that this is not the case, paresis of accommodation being manifest about as soon in many people over forty as in young people.

With regard to cycloplegics his experience, reported already in a paper before the section, was that considerable errors of refraction—sometimes as much as 2 D. of hyperopia and a notable amount of astigmatism—were often latent after the age of forty, and that in spite of most careful precycloplegic testing could not be elicited without a cycloplegic. Whatever glass we gave—and this must be decided by a variety of considerations (certainly not by the cycloplegic examination alone)—he did not see why we should not have the often indispensable information that the cycloplegic gave us.

Dr. G. W. VANDEGRIFT in concluding stated that he always used a cycloplegic in patients under thirty-five years of age and frequently in older people. He does not use a cycloplegic in older people when he is certain that his measurements are correct, both subjectively and objectively by such means as the ophthalmoscope and ophthalmometer. He would rather displease the patient by the use of a cycloplegic and be certain of the exactness of his work than run the risk of prescribing inaccurately.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, LONDON.

The ordinary meeting of the Section was held on March 3, 1920, under the presidency of Mr. W. T. HOLMES SPICER.

Mr. J. H. PARSONS showed a case in which he performed iridotomy. The patient came with **immature cataract**, in which the opacity was chiefly in the posterior cortex. He did the ordinary extraction, with a peripheral button-hole, a simple extraction first, then made a tiny hole in the periphery of the iris afterwards. The patient did well, going out with the eye quiet in ten days. A week later, however, she returned with a hypopyon and a flaring iritis, which he regarded as due to endogenous inflammation. He took her in, and treated her in the usual way, but, in spite of atropine and hot bathing, the pupil became very small, and eventually blocked. He described by means of a drawing the method in which he did the iridotomy, namely, puncturing the iris and passing a Tyrrel's hook in behind the iris, with the idea of coming out of the tiny coloboma at the top. There was a breach of iris, and she was left with an artificial pupil below, which looked like an ordinary pupil. She was left with slight vitreous opacities, but, with correction, her vision was $\frac{6}{18}$.

Several members discussed the case.

Mr. PARSONS also showed a case of extraction of a **dislocated lens.**

Mr. LESLIE PATON exhibited a patient who came to the hospital with certain visual defects but whom he was showing because of an extremely readily excited **vestibular nystagmus:**

it was brought on by the mere pressing of cotton wool into the ear.

He also showed a case of **blue dotted cataract.** He had previously had a similar case. The vision in these cases was usually very little interfered with, and the ophthalmoscope revealed, in this patient, very little opacity. With correction, the vision was $\frac{6}{12}$ in one eye, $\frac{6}{9}$ in the other. All, including Lord Rayleigh, seemed to agree that the blue color was not due to the presence of pigment, but to a refractive peculiarity.

Mr. CHARLES WRAY said there was a family history of the condition in several of the cases.

Mr. BISHOP HARMAN referred to two cases in which there was an association of this peculiarity with night-blindness. Mr. TREACHER COLLINS and Mr. ERNEST CLARKE also referred to similar cases.

Other cases included one by Mr. LESLIE PATON (mass of connective tissue covering the disk) and Mr. M. S. MAYOU (gummata of eyelid).

Mr. W. H. MCMULLEN showed a case of **congenital aniridia,** which was associated with some signs of glaucoma. From the momentary view of the disks which was possible, they seemed to be definitely cupped, and palpation seemed to indicate a raised intraocular tension. He raised the question whether trephining should be done, or some other form of operation with the object of allaying irritation. Or should one wait and note what happened?

Mr. BISHOP HARMAN suggested it might be well to try cyclodialysis for the separation of the ciliary body: the results of this in Vienna had been declared to be satisfactory, and it was said to produce the least interference in an eye in regard to which one was doubtful whether any interference should be undertaken.

Mr. FRANK JULER suggested that a trephine operation might be dangerous in this case, as there must be very little iris tissue left, and in making a trephine hole into the anterior chamber one was apt to wound the lens. He spoke with knowledge of a case in which he saw disastrous results.

Mr. M. S. MAYOU said he had trephined a case of aniridia, and it was satisfactory.

Mr. MCMULLEN also showed a case of **cyst in the outer part**

of the orbital margin of the left eye. He believed it to be a cyst of the orbital portion of the lacrimal gland: it was adherent to the roof of the orbit, and only loosely attached to the tarsus. It did not present behind the lid, as did the ordinary lacrimal cyst.

Mr. TREACHER COLLINS said a feature which helped to confirm the diagnosis of cyst of the lacrimal gland was that when the patient went out into a cold wind the cyst became bigger.

Sir WILLIAM LISTER spoke of a case in his experience in which a cyst of the lacrimal gland went behind the globe. The boy had proptosis of the left eye, which got worse, and the eye was pushed downwards and inwards. He did Kronlein's operation, and after cutting through the periosteum and separating the orbital fat, he encountered a very black tumor, and in proceeding to separate this, it suddenly burst in his face, and the contents practically disappeared. He feared infection at the time, as the lad had somewhat severe acne of the face. But the wound healed up by first intention, and the vision—which had gone down to $\frac{6}{24}$, with contraction of the field—was $\frac{6}{6}$.

Mr. CHARLES WRAY suggested that the cyst should first be punctured, as that would greatly facilitate its dissection.

Mr. JEREMY said he had removed a cyst from the inner side of the upper lid, and it was found to be lined with squamous epithelium and contained mucus. He thought it was a transplantation cyst, and suggested that was the nature of Mr. McMullen's.

Mr. LESLIE PATON discouraged preliminary puncture of the cyst as it might be a cyst of Krause's gland.

A New Test of Color Vision.

Dr. EDRIDGE-GREEN exhibited, by means of the epidiascope, a new series of cards designed to test various forms of color vision defect. The basic idea was a large number of fancy forms which could not be described or memorized by the person submitting to the test, and these were, in succeeding cards, colored in various ways, and large designs wrought in one

color with a varying background. He had not known this test to fail in any single case.

Mr. J. H. PARSONS pointed out the great difficulty of ensuring that every test-set bore identical shades of color: that had been found to be a real drawback in all the published tests. Not only did different editions of the prints vary, but copies of the same edition did so, no doubt largely to the fact that aniline dyes faded, and the progressive penetration of the color into the lithographic stone.

Cobalt Glass in Detecting Astigmatism.

Mr. LESLIE PATON read a note on the use of cobalt glass for testing for errors of refraction, especially small errors in astigmatism, his object being to refresh memories of the use of this glass for the purpose. He gave a demonstration.

In the discussion it was stated that there was likely to be unnecessary trouble in testing if this were employed, as patients would worry unnecessarily over quite trivial errors and bother the ophthalmologist accordingly.

The Treatment of Prolapsed Iris following Perforating Wounds.

Mr. CHARLES GOULDEN read a paper entitled **The treatment of prolapse of the iris following accidental perforating wounds: with a note on the removal of non-magnetic foreign bodies from the anterior chamber.** He said attempts at reposition of the prolapsed iris should be avoided, owing to the risk of infection of the interior chamber of the eye, and because such reposed iris would again prolapse. The patient should not be left with the iris incarcerated, otherwise there would either be delayed healing of the wound, or a weak scar which might bulge under intraocular pressure, or defective vision due to displacement of the pupil, and irregular refraction of the cornea, iridocyclitis, or sympathetic iridocyclitis. If several days had elapsed since the wound was received, the case became much more difficult to deal with. Still, cases could be operated upon and the wound freed from uvea as late as ten days after the accident.

The author proceeded to detail the measures he employed in each type of case, which a condensed report without diagrams could not do justice to.

The paper was very appreciatively discussed by several members.